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# THE MEDICAL JOURNAL OF AUSTRALIA

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VOL. I.—16TH YEAR. SYDNEY, SATURDAY, MAY 11, 1929. No. 19.


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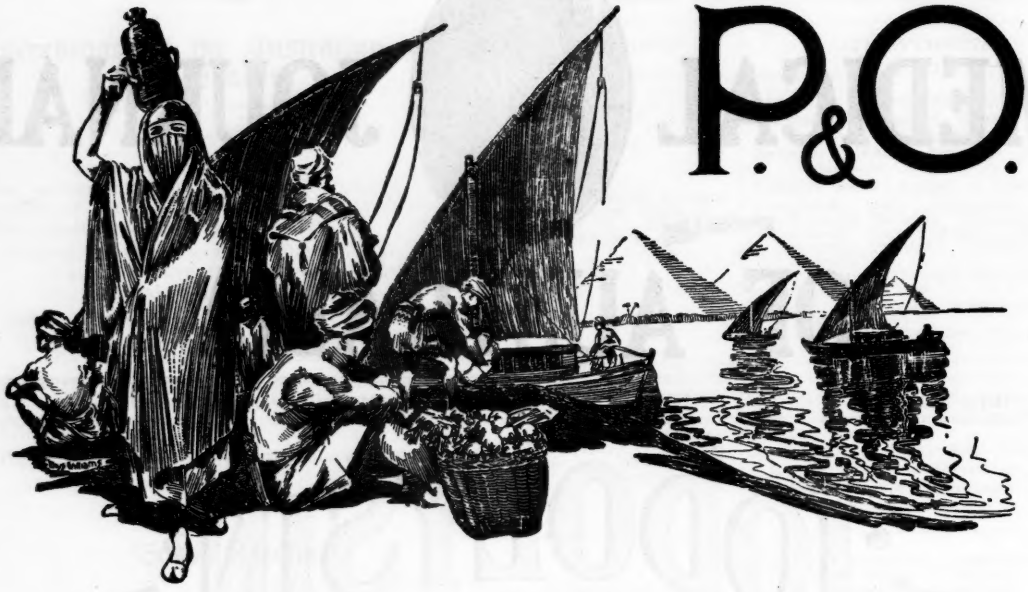

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# THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—16TH YEAR. SYDNEY, SATURDAY, MAY 11, 1929. No. 19.

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# CALCIUM DEFICIENCY IN RELATION TO NERVE DYSTROPHY, ESPECIALLY OF THE AUTONOMIC SYSTEM.

By C. E. CORLETTE, M.D., Ch.M.,
*Consulting Surgeon, Sydney Hospital.*

## Preliminary Note.

THIS paper is intended to be a further contribution to the subject of disease conditions connected with calcium or calcium phosphate deficiency. I contributed a paper on this subject to THE MEDICAL JOURNAL OF AUSTRALIA[1] of March 18, 1928. There was an earlier paper on paroxysmal hæmoglobinuria[2] in the same journal which was also intimately connected with the same subject. I have tried to make the present paper self-contained, as far as possible, and I have therefore repeated some matter as if it were new, rather than refer readers back to previous work which perhaps they have not at hand.

## Grades of Nerve Derangement.

A dystrophy of nervous apparatus may proceed to various lengths. It may advance as far as the destruction of nerve tissue and permanent loss of function. But it may not go so far. In the earlier stages, the change may show itself only by a depression of function, great or small, that is still compatible with quick recovery when the nutritional or toxic or other cause is removed. A conspicuous example is seen in the paralysis of pigeons or other birds induced by lack of vitamin *B*. An acute condition gravely dangerous to life may be recovered from in a few hours if the bird is given an effective dose of the vitamin by administering yeast, or rice polishings.

We can find parallel cases in the clinical group discussed in my paper of March 18, 1928, on calcium deficiency in relation to diseases of animals and of man. In that paper I think I presented cogent reasons for regarding a large number of morbid conditions as really homologous in nature, and due to a deficiency of calcium and/or phosphorus, however brought about. Some cases were associated with deficiency of vitamin *D*, or perhaps with ergosterol deficiency.

Amongst these various homologues were the milk fever and lactational dyspepsia of cows, the post-parturitional paralysis of swine, the cattle disease known as lamziekte in South Africa, the cage paralysis of animals, the rickets and associated paraplegia of monkeys and other animals, osteomalacia and its various nervous accompaniments, some cases described as forage poisoning, certain cases of spasticity and paraplegia in birds, and diseases usually described as ergotism and lathyrism.

In the incoordination, spasm, or paraplegia manifested in these conditions, very quick recovery may be possible in some cases, as is shown by the seemingly miraculous effect of pumping up the udder with air in cases of milk fever. But in some there may be a certain amount of residual paralysis that is slow in recovery. In the cases of quick recovery, the damage may be presumed not to have gone beyond the functional stage, and recovery follows the stoppage of the drain of calcium phosphate. In the others there has been some disorganization of nerve tissue that has gone past the merely functional. The results of Hedinger's histological examinations of the cord and peripheral nerves in fatal cases of lamziekte explain what takes place.

In parentheses, let me say that in my last paper there was no space to spare for references to all investigators. There is an especially good paper by H. Wehrbein[3] who found degenerative changes in the peripheral nerves of swine suffering from what is evidently a homologous type of disease. It is well-known in pigs, both in the growing stage and as a post-parturitional development, the former corresponding to the paralysis accompanying rickets, and the latter to that of milk fever or lactational dyspepsia of cows. Hart, Miller, and McCollum[4] recorded about the same time degenerative cord changes (peripheral nerves seem not to have been examined) in pigs suffering from paralysis which they considered (at that time, at all events) to be caused by some toxic factor present in wheat grain. I have no doubt that this paralysis belonged to the same group as the others I have discussed. On this point refer to the discussion of lathyrism in my last paper. No one seems to have recognized that calcium privation will cause paralysis, and that through its control of calcium metabolism vitamin *D* is as truly an antineuritic vitamin as is vitamin *B*. I think I have proved this abundantly in my last paper.

For convenience, I will refer to the type as the lamziekte, or calcium-lack type, of nervous phenomena, whatever the various clinical names which disguise it.

The disorder of nerve function manifested in the lamziekte disease-type tends to be very wide, more or less affecting all parts of the nervous system. Although the motor apparatus suffers most prominently in this group, as it does in the beri beri group also, yet there is evidence that the afferent nerves do not escape, for in some victims there has been noted a deficiency of skin sensibility. There is also evidence of emotional excitability, such as timidity or irascibility, or cerebral torpor and dulness, and sometimes of delirium, all of which we can interpret as due to derangement of cerebral function. In some cases there are general convulsions of cerebral (epileptiform) type, though tonic spasm of the tetany type is more frequent. In bad cases there is amaurosis, the animal appearing to be blind, signifying derangement in some part of the optic apparatus. Vertigo is frequent, apparently indicating some disturbance of cerebellar function. Polypnœa and tachycardia are reported in some cases. The very common constipation may indicate interference with the motor innervation of the bowel.

## Parallelism with Lead Poisoning.

Signs are not wanting that there is a tendency to derangement of the nervous apparatus in the

lamziekte type of nerve disorder, comparable to and quite as wide in its effects as that of lead, and it is possible that minor degrees of it may be much more common than most people imagine.

We may call to mind here some of the ways in which lead poisoning may be manifested. A prominent and often early sign of nerve implication in man is a paralysis or paresis of the limbs, chiefly involving the extensors, and causing dropped wrist. In quadripeds this would possibly be represented in mild cases by "knuckling over" during locomotion, and in other cases by "walking on the knees" or "walking on the wrists." In lead poisoning, the paresis is due to peripheral neuritis, but locomotor difficulties could also arise from tonic flexor contraction dependent on central nervous changes. I should imagine that in animals most cases of knuckling over, walking on the knees, or walking on the wrists, or toe-walking (the last is seen in rats and dogs) would be due to spastic difficulties, for in anything but very slight paralysis it must be very difficult to walk at all.

As another occasional manifestation of peripheral neuritis in lead poisoning, Law,[5] Thomasson, and Beckmann and Schmidt, the last three being cited by Hutyra and Marek,[6] describe paresis of the recurrent laryngeal nerve—indicating implication of the vagus nerve. Paresis of the recurrent laryngeal nerve is the cause of what is called "roaring" in horses.

Lead poisoning also produces effects on the intestinal apparatus. It causes obstinate constipation and severe intestinal colic. This appears to indicate a nerve disorder affecting the peristaltic movements of the intestine. Disturbances of function of the spinal cord nuclei occur also as a result of lead poisoning. Signs of lead poisoning may occur also in connexion with the visual apparatus. Amaurosis occurs occasionally; night blindness (the so-called hemeralopia) has often been reported; other eye complications are retinitis, retrobulbar neuritis, and optic atrophy. Signs of cerebral damage also occur. There may be epileptiform convulsions, delusions, delirium, mania and permanent insanity.

But there is still more, for the effects of lead poisoning extend beyond the nervous system. For instance, it may be a cause of anæmia, abortion, nephritis, and gout.

### The Vagus Nerve.

Now, if the disturbance of nerve nutrition in this type of deficiency disease be really so widespread as to be comparable in certain directions with those manifested in lead poisoning, what about the other directions which we have not yet explored? Some of the nervous manifestations met with from time to time in lead poisoning indicate implication of the vagus nerve. Partial paralysis of the recurrent laryngeal is one. Does vagus disorder begin and end with implication of the recurrent laryngeal branch? Are there ever any symptoms which on reasonable interpretation seem to point to other regions of distribution of the vagus? Let us see what these other regions include. The vagus not only conveys fibres supplying the larynx, but it conveys autonomic fibres supplying the trachea, bronchi, lungs, heart, stomach, duodenum, liver, pancreas, spleen and kidneys. Certain of its fibres join the ganglia of the solar plexus; these fibres pass through the ganglia and become associated with other fibres derived from the sympathetic, and hence in connexion with the spinal cord. These vagus fibres supply the whole length of the small intestine and the greater part of the large intestine. The vagus supply of the stomach and intestines belongs to the viscero-motor part of the autonomic system. The muscular coats of the stomach and intestine have a double set of nerves, each set exercising a different function, one excitatory and contractile, the other inhibitory. Both sets act in coordination. The inhibitory fibres are those derived from the sympathetic. Stimulation of the vagus, therefore, produces contraction of both circular and longitudinal coats of the stomach and intestines, whereas stimulation of the sympathetic fibres inhibits contractions produced by stimulation of the vagus. There is also evidence of a local mechanism, since contraction and peristalsis may occur after section of all the nerves. Furthermore, there is, besides the nerve control, a chemical control through hormones, the adrenal hormone acting similarly to the stimulus of the sympathetic and causing dilator effects, while the secretion of the infundibular portion of the pituitary acts as a constrictor. The autonomic nervous system has a similar type of control over secretory function, the pressor fibres being derived from the vagus, the inhibitory from the sympathetic. On the other hand, in regard to vasomotor control, it is the sympathetic that supplies the constrictor fibres, and the vagus the dilator fibres.

The vagus is also the path of afferent nerves from the stomach, and apparently also from the intestine and other organs, though the afferent impulses are not ordinarily evident to consciousness. It is by this means that the sense of fullness of the stomach is conveyed, and it is evidently by the same means that vertigo is caused under certain conditions, and in animals that vomit, the afferent stimulus provoking that act would travel by the same path.

### Paralysis or Paresis of the Vagus.

Now let us see what happens when the vagus is paralysed, so far as the œsophagus, stomach, and intestines are concerned. Section of the vagus causes paralysis both of its motor and sensory function. The result of stomach paralysis is that its expulsive power is lost, and nothing passes on into the intestine. Secretory function would also be interfered with, but I will not discuss that here. As regards ruminants, F. Smith[7] quotes Ellenberger as having shown that section of both vagi caused paralysis of the œsophagus, rumen (first stomach), and reticulum (second stomach), followed by tympany, or distension, of the rumen. The omasum (third stomach) is not affected, since it is said not to be innervated from the vagus, but

from some as yet unknown source. As regards the horse, Smith quotes Chaveau as having found that if both vagi were divided in the neck, and the animal fed, the stomach and whole length of the œsophagus frequently became greatly distended with food in consequence of loss of motor power, and he conjectured that the sense of repletion is lost in such cases, this accounting for the animal's continuing to eat. It seems reasonable to assume that this defect of muscular contraction is continued beyond the stomach into the intestines, though specific information is not given. It seems to me that we are on strong ground in inferring that defective muscular tonus in the stomach and intestines is producible by relative inadequacy far short of complete paralysis of the controlling mechanism of the vagus, and since normal afferent, as well as normal efferent stimuli are necessary, the regular coordination of motor and secretory function with requirements is likely to be disturbed. If this be so, we can imagine as a result, not merely a simple dilatation of the stomach, but accumulation of gastric contents within it, causing what is in animals described as "impaction," and fermentation of the contents causing what is called "tympany," the total result being extreme distension. In the intestine we might also get tympanic distension and (since we are not dealing necessarily with paralysis, but incoordination and inefficiency) irregular spasm producing colic. And there would usually accompany it an obstinate constipation—perhaps varied by diarrhœa if local stimuli were sufficient, as in cases of infection. An outward manifestation of loss of tone in the intestine would be "pot-belly."

### Bulbar Paralysis.

In introducing the present discussion, I referred to lead poisoning for the purpose of getting and following suggestions as to lines of study. I mentioned Law, Thomassen, and Beckmann and Schmidt as authorities for the statement that laryngeal paralysis was in some cases a result of lead poisoning. The last three authorities were given as quoted by Hutyra and Marek.[6] Let me now give the full quotation from Hutyra and Marek:

> Thomassen proved experimentally beyond a doubt that in the horse lead is capable of causing degeneration of all the branches of the vagus, the degeneration being most pronounced in the terminal portion of the left recurrent nerve. The observations of Beckmann and Schmidt yielded the same results; animals working in the neighbourhood of lead works are affected in large numbers.

Under the caption "Paralysis of the Vagus," Hutyra and Marek again quote Thomassen as having adduced experimental evidence that in the horse chronic lead poisoning causes extensive paralysis of all the branches of the vagus, and then they add to this the remark, "the various species of *Lathyrus* appear to be capable of producing a similar condition." But, if I am right, lathyrism is not a toxic disease, but a deficiency disease, the same in fact as that seen in lamziekte, milk fever, and their other homologues. Hutyra and Marek's statement would then mean that this deficiency disease is capable of causing extensive paralysis of all the branches of the vagus.

Hutyra and Marek further remark that bulbar paralysis is generally followed by paralysis of the recurrent laryngeal nerve. In my last paper I quoted from a text-book description which showed that paralysis of the larynx occurred in certain bad cases of milk fever, in connexion with other symptoms indicating bulbar paralysis. What is liable to cause bulbar paralysis, is liable to affect also the recurrent laryngeal nerve. And the same noxious influence that will affect only the recurrent nerve in one case will do much more in cases of greater severity. It may, for instance, become a cause of bulbar paralysis. This is entirely reasonable in the light of general pathological and clinical experience. Again, what affects the centre, may on occasion affect, or seem to affect, the peripheral part first or independently, and this would furnish cases of peripheral recurrent nerve paralysis. The further point that this individual branch of the vagus stem may not carry a monopoly of the detriment has already been considered, and will be considered again.

It may be said that these ideas are mere exercises of imagination. They are. But in the present state of our ignorance, that is almost all that we can do. Still, they are not mere random and irrational vagaries. We must use the known as points of departure, and these point as finger posts to lines along which we can look. Present-day knowledge of neurology certainly gives sanction for the entertaining of such ideas.

It is unquestionable that bulbar paralysis is manifested in serious cases of lamziekte, as also in serious cases of milk fever. Mild symptoms—difficulty of prehension of food with difficulty of swallowing—occur in bran disease (osteoporosis, osteomalacia) of horses. It is also unquestionable that in lamziekte, at all events, there is clinical and histological evidence of peripheral neuritis, and also of pathological changes in the spinal cord. I have offered a theory of pathogenesis to account for this widespread damage to the nervous system.

We do not always find specific mention of laryngeal paralysis in clinical descriptions, but there are reasons why, if it occurs, it should not be obtrusively evident, for the clinical signs of partial paralysis are usually only made obvious on unusually forced respiration such as is provoked by violent exercise, a test inapplicable in a paralysed animal. Even in cattle whose locomotion is unaffected, there is not usually such violent activity as operates so often to reveal it in the horse, a much more active animal, by the symptom of "roaring." It is in forced inspiration that the glottis requires wide opening, and a paralysed abductor makes the wide opening impossible. But it is obvious that there are degrees of paralysis. It should also be emphasized that in the beginning there may be only a functional disturbance from which it is possible for

recovery to be rapid, just as there is in the beri beri group.

I have suggested that a biochemical disturbance able to cause central damage in which laryngeal paralysis is produced, may in minor cases be a cause of localized peripheral damage. If that is so, we should be prepared to find that the disturbance which causes lamziekte and its homologues would, on occasion, produce laryngeal paralysis as the sole apparent symptom. However, it does not necessarily follow that the central part is in no way affected because, at the moment, the only apparent change is manifested in the peripheral part.

Now, do these hypothetically possible cases occur as actual phenomena? Do we find clinical evidence affirmative?

### Laryngismus Stridulus.

One's attention is here attracted to the phenomenon of *laryngismus stridulus* in children, which is described as almost invariably confined to those suffering from evidence of rickets. I have never seen or heard of a case in Sydney, but according to writers, it is common in Britain. In *laryngismus stridulus* the clinical picture is one of paroxysmal spasm of the laryngeal muscles. It is therefore not itself a paralytic phenomenon. However, it is quite obviously specially localized. It involves selectively the parts supplied by the laryngeal branches of the vagus. It is distinctively a nervous affection. But this is not the only evidence of disordered nerve function exhibited by these or other rhachitic children. They exhibit the symptom known as spasmophilia, in which there occurs an increased electrical and general excitability of the motor nerves supplying the voluntary muscles. This is evidently not a sign of increased well-being and better functional efficiency, but of the reverse. It is in fact regarded as a sort of latent tetany, and it would appear that the laryngeal affection is a special development of this general phenomenon. Furthermore, in many of these children this condition passes over the threshold, and frank tetany is the result. It should be noted that in the milder forms of tetany the extremities alone are affected, but in the severe forms the muscles of the trunk are also involved. That is to say, in the milder forms the manifestation is confined to the parts furthest from the nutritional centre of the nerve unit. Sometimes these children also exhibit general convulsions, which can only be interpreted as due to disorder of the central portion of the nervous system.

We do not find mention of paralysis of the limbs in connexion with rickets in man, but we do in animals. It will be remembered that this was noticed, as well as incoordination, tetany, and convulsions, in Mellanby's rhachitic puppies.[8] Paralysis was also a conspicuous symptom in the rhachitic monkeys and *carnivora* in the London Zoological Gardens, described by Bland Sutton.[9][10][11][12]

There is, however, another phenomenon associated with rickets and with *laryngismus stridulus* in children, and that is "pot-belly." Pot-belly is likewise observed in rhachitic animals. It occurred in Chalmers Watson's meat-fed rats, accompanied by accelerated and noisy respiration.[13] So also is it a characteristic of animals suffering from deficiency of vitamin *B*. It was noticed in the animals suffering more or less multiple deficiencies in McCarrison's experiments,[14] to which I shall give more particular attention directly.

### Roaring in Horses.

I have already referred to the symptom known as "roaring" in horses. Apart from transient swellings, and the like, it is a symptom nearly always indicative of some degree of paralysis of the left abductor muscle of the larynx, and this receives its nerve supply from the recurrent laryngeal nerve. It is a peculiar sound audible only on inspiration, and it is said to be produced by the incomplete separation of the vocal cords through deficient action of the abductor, and a simultaneous sucking downwards of the left arytenoid cartilage towards the glottis by the inward current of the air. In quiet breathing there is no abnormality noticed. It becomes evident only on violent inspiration, and it is frequently tested for by galloping the animal. It has been reported as occurring in dogs used for draught purposes, but it is characteristically a malady of the horse. It is popularly believed to be hereditary. The same belief exists about periodic ophthalmia, another malady of the horse. I have not yet put forward any discussion of periodic ophthalmia, but I would say here that my studies go to suggest very strongly that the heredity is merely the inheritance of a similar environment or system of feeding. And it seems to me that the same applies to roaring.

According to Hutyra and Marek,[6] roaring is:

> Exceedingly common in horses in England, France, Hanover, Holstein, Austria, and Hungary, and in countries in which horses are stall-fed and seldom pastured.

They go on to say:

> In the Argentine and in South America generally, in Egypt, India, Arabia, and Australia, the disease is rarely met with.

I am not sure about Egypt and India, but as for the rest, he picks out countries where pasturage is within the reach of most animals all the year round.

Roaring appears to be specially common in race-horses, as is shown by the following passage from Law:[5]

> The relatively great prevalence of roaring in the thoroughbred English race-horse at home (5 per cent. in the army in 1888) and in descendants of this race in other countries implies a special cause connected with conformation or usage. The spare habit of the body, the lack of fat, and the violent exertion demanded of the animal have been adduced in explanation.

Law conjectures that the cause may be found in the great development of the heart, which produces mechanical interference with the left recurrent nerve. Hutyra and Marek remark, however, that investigations appear to support the view that in the majority of cases infective diseases are to blame

for the causation, and that experience shows that the disease is a common sequel to strangles, pneumonia, or infectious sore throat. If that is so, one begins to ask oneself why race-horses should be so particularly liable to contract these infectious diseases. Is it because of a lowered resisting power? Or if they are not especially liable to the diseases themselves, one wonders why roaring should be more liable to follow those diseases in the race-horse than in other horses. Hutyra and Marek also state that in many cases rheumatism is supposed to be a cause. This supposed relationship to rheumatism is interesting, for rheumatoid maladies of horses and cattle appear to occur in peculiarly frequent association with what I have identified as the calcium, or calcium phosphate, deficiency group of homologues, so frequent that it must be more than accidental. Periodic ophthalmia of the horse has the same peculiarity.

While roaring appears usually to continue as a chronic or permanent affection, it is said to recover spontaneously sometimes as a result of "change of climate," which, I would remark, might often mean and include much more than mere climate. Other changes of environment, not climatic, but not infrequently liable to involve dietary changes, are also said to produce improvement sometimes, as, for instance, in stallions when transferred to stud.

It is not necessary to suppose that when the symptom is a permanent one, the originating cause of the lesion is also equally persistent, for if the muscle fibre, or the nerve centre, or part of it, has become irretrievably ruined, paralysis would then remain, despite cessation of the originating cause.

In looking for the originating cause of roaring, one's attention is arrested by the extraordinary and striking fact that this malady is said to be specially common in race-horses. In my last paper[1] we became acquainted through Hutcheon[15] with another malady to which race-horses have, in some stables, or places, or under some systems of management, been peculiarly liable, and that is osteoporosis. Hutcheon was disinclined to believe that osteoporosis could be the result of a faulty dietary, because, as he remarked, "the feeding was of the best," and he therefore favoured a theory of infection. However, these horses tended to recover when given a certain kind of "change of climate," but the change of climate included a change to fresh green pasture. A statement that the feeding in a racing stable was of the best simply means that it conformed to orthodox traditional ideas as to what was "best." In regard to the feeding of race-horses, it may be alleged that long experience has shown that a certain system is "best." That only means that it is as good as practical men have been able to make it, not that it is flawless. But "practical" men have their limitations. We may readily admit that it is excellent for its immediate purpose. But is it perfect in a general sense? It is conceivable that it may be efficient for its purpose and yet be rather finely balanced in some ways between surplus and deficiency. And as to "experience," it must be pointed out, as I have shown, that osteoporosis is part of that experience. Periodic ophthalmia, which is said to be particularly frequent in race-horses, would appear to be another part of that experience. In discussing that disease, Law[16] remarks, with specific reference to race-horses, that the period of training is the most common occasion for the attack "partly because he is made to exchange the cooling succulent grass for the stimulating ration of hay and grain."

Is roaring another part of that experience? It would seem so. It would be merely the attitude of a shut mind to argue that the system of dieting being "perfect," diet cannot possibly be involved in the production of roaring, and, consequently, that another cause must be looked for.

I think I have shown[1] that the customary dietary of stall-fed horses is not likely to be very high either in calcium or in vitamin *D*, and that we should not expect the fat of these animals to be very rich in vitamin *D*, perhaps not any richer than is the fat of grain-fed pigs. An unrestrained voracity of appetite (polyphagia) might overcome any real deficiency. I showed the important protective function of polyphagia in my last paper. The scantiness of reserve fat in a race-horse under training would presumably reduce reserve stocks of vitamin *D* to the lowest possible point, though adequate sunlight exposure would probably make this fact relatively unimportant. Nevertheless, if the customary dietary is such that it is near the threshold of insufficiency in calcium, it becomes specially necessary to preserve the power of dietary assimilation of calcium at an optimum point, and the same applies to the power and facility of using the calcium reserve in the bones, but under the circumstances described it is doubtful if this efficiency is always adequately maintained.

### The Calcium Content and the Phosphorus : Calcium Ratio in Different Samples of Hay.

The ratio of phosphoric acid to lime (phosphorus pentoxide to calcium oxide) in samples of oat hay varies a good deal. In samples analysed by H. Ingle[17] in South Africa, the ratio was as wide as 1 : 0·23 in one set, and as close as 1 : 0·65 in another from a different district. In another hay (Natal blue-grass, not oats), the ratio was 1 : 1·68. But ratios quoted from Wolff were 1 : 2·47 for European meadow hay, and 1 : 4·76 for lucerne (alfalfa). In European oat grain the ratio was 1 : 0·16. It will be seen that the South African oats gave a ratio more comparable to that found in grain than to that found in the European meadow hay. The wide ratio means that most of the phosphorus cannot be stored in the bones as calcium phosphate, because there is not enough calcium to combine with it and fix it (law of the minimum). The South African oat hay showed, moreover, in most samples a considerably lower figure both for phosphorus and calcium than those shown by quoted analyses of European oat hay.

In certain feeding experiments at the Agricultural Experiment Station of the University of Wisconsin recorded by Hart, Steenbock, and Humphrey,[18] one sample of oats grown on an alkali soil, that is, in older and more homely English, a brack soil, at the farm contained 0·84% of calcium oxide, but in a set of earlier experiments in which bad results were obtained, the oat hay, which was grown elsewhere, contained only 0·47% of calcium oxide. To a horse, the calcium oxide content of one of these samples is equivalent to that contained in twice the amount of hay of the other sample. One may see in this once more how polyphagia would protect an animal in such circumstances. Surely I am right in calling polyphagia the manifestation of a protective physiological instinct? The possibilities involved when the horse is already near the threshold of deficiency, and when indulgence in polyphagia is for any reason impossible, are so obvious as to need no more words.

It seems, therefore, that several outwardly different clinical manifestations of disease may contain a common factor of causation. Notwithstanding this, there are some difficulties in accepting this common factor as the sole one operative, for why should the manifestation be in one case chiefly, or apparently, osteoporosis, in another ophthalmia, and in another roaring? The acceptance of a deficiency theory does not at present remove all difficulties. At the same time, the continued existence of such unsolved questions does not block the way and make all efforts at interpretation sterile. After all, we do find permutations and combinations in the symptomatology of disease conditions otherwise connected by some underlying, but unseen, common factor. We find a special tendency to affection of one nerve group in *laryngismus stridulus* of children. Why not other nerve groups just as often? Why may osteoporosis exist in rickets without ophthalmia and without *laryngismus stridulus*? Why do not rhachitic children become paralysed like rhachitic monkeys? Of questions that may be asked there is no end. It was with careful purpose that I introduced the subject of lead poisoning at the beginning, because of its protean manifestations. Why does lead sometimes seem to cause colic only, sometimes eye complications only, sometimes "dropped wrist" only, or even gout only? I do not say that it really confines its damage to one channel, but that it may seem to, which is another thing.

### Disorder of the Visceral Autonomic Nerve Apparatus.

We have seen that in certain conditions of impaired nutrition, or of toxic interference, nerve tissues manifest disorder of function, and sometimes gross pathological changes. Amongst these conditions one may particularize deficiency of vitamin *B* (beri beri), deficiency of vitamin *D*, deficiency of calcium and/or phosphorus (lamziekte, milk fever, cage paralysis), and lead poisoning. I think I have shown that the tendency is very widespread within the nervous system, and that sometimes the incidence may fall more on one part than on another. The recurrent laryngeal branch of the vagus is one of these parts, as is shown by a tendency either to muscular spasm (*laryngismus stridulus*) or to a partial paralysis, especially abductor paralysis. I have argued that there is an imminent possibility that other parts and other functions of the vagus may, in the same or in other cases, suffer some degree of detriment, and I have outlined some of these other parts of distribution, and their function. When the autonomic apparatus is in disorder, we can to some extent point to one part and say it is included, but we cannot at the same time say the coordinated parts are not included. On the contrary, we may be sure that all parts are liable to suffer. As an illustration of our ability, at times, to point specially to one part and say it is included, we may take the case of acute fulminating beri beri in man. It is said that in the most acute cases the patient may suddenly fall down dead, and we may remember that similar catastrophes are seen after diphtheria. I have never seen such cases as those mentioned, but within my own experience in beri beri I have several times seen death with signs of urgent cardiac distress occur within a few hours of the apparent commencement of the attack. This points (at least, that is the general opinion) to a severe and special implication of the vagus nerve mechanism of the heart.

We are indebted to McCarrison[14] for a number of extremely important observations on the visceral lesions that occur in pigeons, guinea-pigs and monkeys subjected to dietary deficiencies, and a description of some of these will help us considerably in our present study. In McCarrison's own discussion of the experimental results he did not consider the possibility that deprivation of vitamin *D* or of calcium or phosphorus might be also a cause of nerve disorder. His attention in that respect was fixed on vitamin *B*, as has been the case with all other observers up to the present. However, the point as to whether nerve disorder could have been in some cases contributed to by other deficiencies than that of vitamin *B* is really immaterial to the purpose at present in view. The really important thing is the exhibition of what is found in a disease in which nerve degeneration is a prominent feature, whatever the cause of the degeneration.

All the same, in looking at the description of pathological appearances, one must carefully remember that coexistence with nerve degeneration does not necessarily mean secondary to nerve degeneration. It is probable that much damage comes from other directions. A direct detriment to cellular nutrition in general might be caused through absence of elements necessary to processes of cell nutrition. Again, there is a probability of metabolic poisons entering the circulation, or forming locally, through derangement of normal detoxicative processes. Still again, there is the factor of infection, a result of diminished resisting power of the damaged tissues. However, in spite of all

these possibilities of complication, the fact will still remain that if nerve apparatus is injured, nerve function must suffer.

### McCarrison's Observations on the Gastro-intestinal Tract in Deficiency Disease.

Bearing all the above possibilities in mind, let us now proceed to note some of McCarrison's observations on the gastro-intestinal tract of polyneuritic monkeys.

McCarrison says that, in general, the disorders resulting in this situation from the use of the deficient foods were:

1. Congestive, necrotic, and inflammatory changes in the mucous membrane; sometimes involving the entire tract, sometimes limited to certain areas.

2. Degenerative changes in the neuro-muscular mechanism of the tract, tending to dilatation of the stomach, ballooning of areas of small and large bowel, and probably also to intussusception.

3. Degenerative changes in the secretory elements of the tract—of the gastric glands, the pyloric glands, the glands of Brunner, the glands of Lieberkühn, and of the mucous glands of the colon. On this he remarks: "These changes are such as must cause grave derangement of digestive and assimilative processes."

4. Toxic absorption from the diseased bowel as evidenced by the changes in the mesenteric glands.

5. Impairment of the protective resources of the gastro-intestinal mucosa against infective agents, due to hæmorrhagic infiltration, to atrophy of the lymphoid cells, and to imperfect production of gastro-intestinal juices. "This impairment (he says) results not only in infections of the mucous membrane itself, but permits of the passage into the blood-stream of micro-organisms from the bowel."

The stomach was invariably greatly dilated, and its walls much thinned, and quite empty. Ecchymoses were frequently present, especially at the pyloric end. Ulcers were sometimes found in the pyloric area.

The small intestine was always greatly thinned, either throughout its whole extent, or more particularly at certain areas. Ballooning of the small bowel was present in a very large proportion of cases. The ballooning was usually in the ileum. The ballooned areas were as a rule multiple, and of varying extent. The ballooning was not due to accumulation of intestinal contents, but to excessive thinning and atony of the bowel walls. Intussusception was very frequent. It was present in ten out of thirteen rice-fed monkeys. The invaginations were usually multiple, the upper part being invariably invaginated into the lower. McCarrison writes:

> The majority of these intussusceptions were no doubt manifestations of the death agony. In some, however, although no inflammatory changes had occurred, considerable constriction was present, the invaginated bowel being then much engorged, and sometimes of a reddish-brown hue. It is concluded that such invaginations occurred at least several hours prior to death. I am disinclined to regard them as invariably agonal in origin; agonal intussusceptions are said to be usually ascending, those in the present experiment were invariably descending. It appears probable that the changes in the neuro-muscular mechanism of the bowel . . . were concerned in their origin.

Subserous congestion and ecchymosis, and also submucous congestion and ecchymosis, with necrotic changes in the mucous membrane, were very marked features. They were usually more pronounced in the duodenum than in the stomach. The changes frequently, but not always, extended to the jejunum and ileum.

The large intestine showed congestion and subperitoneal ecchymosis, ballooning, and thinning and partial disappearance in places of the longitudinal bands of muscle. As a rule there were changes in the mucosa indicating intense colitis, but without ulceration. Amœbic dysentery was present in a number of these cases. The amœbic dysentery was certainly induced by the lowered nutrition and lowered resisting power of the intestine. In passing, it may be remarked that this observation of McCarrison is very important, and provokes a strong suspicion that epidemic amœbic dysentery may oftentimes be, to a large extent, a deficiency disease.

Examinations of sections of bowel under the microscope revealed correspondingly serious pathological changes. The muscular coats were found to be in a state of atrophy, and this was extreme where ballooning had occurred. An important observation was that in all parts there were degenerative changes in the myenteric plexus of Auerbach. The ganglionic cells were swollen and the cell membrane indistinct or wanting. The nuclei had often disappeared, or existed only as fragments of chromatin. Ganglia were sometimes encountered in which no cell contained a normal nucleus. As McCarrison remarked: "There can be no doubt that such changes would impair the neuro-muscular control of the bowel."

### "Meningo-Encephalitis."

There is a remarkable symptom complex, said to occur most commonly in horses, and attributed usually to forage poisoning. It is also described in veterinary literature under the names of encephalitis, meningo-encephalitis, and cerebro-spinal meningitis. In this condition there is clear evidence that the glosso-pharyngeal and vagus (ninth and tenth) nerves are functionally deranged, in other words, there is bulbar paralysis. We have seen that symptoms of bulbar paralysis are also observable in some cases of milk fever, of lamziekte, and of bran disease (osteomalacia). In my last paper[1] I referred to a disease described by Gilruth in New Zealand, and thought to be due to ergot. Let us note here that Gilruth's paper[19] bore the caption "Meningo-encephalitis (Stomach Staggers) in Horses and Sheep." I suggested that ergotized forage had nothing to do with it, and that it was due to calcium deficiency. But as we shall see presently, there is a true forage poisoning, of which the symptoms resemble botulism very closely. It

is evident that the differential diagnosis of animal diseases with more or less similar nervous symptoms is very difficult, and an examination of the literature makes it certain that diseases really quite different are sometimes classed together by able and experienced men.

### Forage Poisoning.

Gilruth[20] has described a series of cases of forage poisoning, and we can gather from his paper an idea of what occurs in the horse when the vagus nerve is functionally disordered. The toxic material was traced to a certain sample of fodder.

> A horse had died after eating this, and an autopsy was made. The knacker who came to remove the body was kept waiting till the autopsy was finished, and while waiting noticed a nosebag half full of feed. Not knowing that this was some of the material under suspicion, and without asking permission, he gave this to his own horse. In six days the knacker's horse was also dead, after an illness similar to that of the first horse. Experiments made with the fodder—bran and oaten chaff—given to cattle and horses all led to death, the symptoms, however, being not in all respects the same in cattle and horses. Horses became paralyzed in their limbs before death, whereas cows did not.

But the main reason why I have recalled Gilruth's cases to notice is this. They show very clearly that one of the symptoms of vagus nerve disorder is colic. This was noticed both in cows and horses, and in horses the symptoms were extremely severe. In some cases the first symptoms were those of a hyperacute and sudden attack of colic. The animals became unable to swallow. In some cases when the animal succeeded in swallowing a little food, it was found after death lying in the thoracic part of the œsophagus; food was also found lying in the stomach. There was obviously bulbar paralysis.

We may expect that in bulbar paralysis of less severity, food could be swallowed a little better, and would reach the stomach, but the stomach, having diminished motor power, would be unable to empty itself, and food would accumulate in it, distending it, and producing the condition called "impaction." In ruminants, the stomach to became impacted in this case would be the rumen, or paunch—the first stomach. But whether the stomach were full or not, food lying stagnant therein would ferment and generate gas. This would distend the organ, producing gastric tympany. In the intestine there would also be atony, gaseous distension, and stasis. In ruminants there would be a tendency to distension of the gall bladder through loss of contractility. Horses have no gall bladder.

As McCarrison himself pointed out, the deficiencies in his animals were multiple. For instance, in monkeys fed on autoclaved rice there would not only be a deficiency of vitamin *B*, the so-called "antineuritic" vitamin, but just as much would there be deficiency of the three fat-soluble vitamins, *A*, *D*, and *E*, the antiscorbutic vitamin *C*, and deficiency of calcium, iron, phosphorus, chlorine, sodium, and other elements.

In the face of the array of evidence in the paralyses of Mellanby's rhachitic puppies,[8] and in the paralyses of rhachitic rats, osteomalacic fowls, and the rest, I doubt if anyone would be bold enough now to maintain that the paralysed and rhachitic monkeys and carnivora of the London Zoological Society's menagerie described by Bland Sutton[9][10][11][12] were merely suffering from a combination of rickets and beri beri. Deficiency in vitamin *B* is probably one of the least likely of deficiencies in the food of those animals. Their paralyses were not signs of deficiency of that particular "antineuritic" vitamin. They were suffering from the same symptoms as were Mellanby's puppies and the rest. Whatever it was that caused rickets and paralysis in the puppies, that was enough to cause, and was the cause, of the same in the menagerie animals. I have already suggested what that cause is likely to be.

Bland Sutton particularly remarked on the size of the stomachs revealed at autopsies on these rhachitic animals. He reported that the stomach was often dilated so as to be twice or thrice as large as normal. In one case[11] (a spider monkey), he relates that the abdomen was so much enlarged as to give one the impression that the distension was the result of a gravid uterus. There was an enormous dilatation of the stomach, which extended from the diaphragm to the *symphysis pubis*.

Do we see in these cases, amongst other things, reasonable evidence of vagus nerve disorder? I think so. What else shall we say? If this be so, and if my theory of pathogenesis be right, we have acquired evidence that, besides the cerebro-spinal nerve apparatus, the autonomic apparatus also is liable to become disordered by deficiency of calcium and/or phosphorus, or by deficiency of vitamin *D*.

I have already suggested that the customary dietary of stall-fed horses may be rather finely balanced between surplus and deficiency, may be in fact somewhat close to the threshold of deficiency on the side of vitamin *D* and calcium, so that a comparatively slight and almost unnoticeable change in circumstances could precipitate an attack of illness. Now, supposing that the illness took the form of an attack of colic, or of impaction, or of acute tympany of the stomach, the apparent and immediate cause would be considered as in itself sufficient to account for it, coupled with the further consideration that the horse is "by nature" extraordinarily prone to colic, acute tympany of the stomach, and so on. That begs the question. Why is the horse so liable? Is it really horse nature? Why do not donkeys suffer in the same way? I have mentioned osteoporosis, ophthalmia, and roaring as items of experience for horsekeepers that may possibly be causally connected with their methods of horsekeeping. Is the tendency to colic and tympany another of these contributions to experience? As to volvulus, which is relatively common in the horse, I note that F. Smith[7] says that "there is no doubt the chief cause of this trouble is tympany." That would be intestinal tympany, like that which McCarrison described in monkeys.

### Dilatation and Impaction of the Stomach.

As regards impaction of the stomach, that is, accumulation of food which distends it and cannot

be propelled onwards, I find it extremely interesting to note that, in addition to attacks precipitated by over-eating of young succulent grass, and the like, in animals long deprived of it, there is another way that is very suggestive. E. W. Hoare[21] quotes C. Reeks as noticing that impaction occasionally developes during the winter months when the provender has consisted only of dry corn and chopped wheat or oat straw. According to Reeks "when one horse out of five or six fed on the same diet suffers from an attack, the explanation is that this animal is a notoriously greedy feeder." Now, I ask, why is he a greedy feeder? It seems to me that my explanation of polyphagia, given in my last paper[1] exactly fits such a case, and provides a biological reason for the greedy feeding. It is to get some element or elements only poorly represented in the corn and straw. But apparently, some animals fail, after all, to get the necessary supply, and as one of the consequences there is some functional inadequacy of the vagus nerve and gastric muscle. Then comes impaction. The polyphagia has failed to protect, because the minimum has not been reached, despite all efforts.

Just here, I would like to make use of a paper by D. Hutcheon[22] based on South African experience. We may observe first that he gives his paper the title "Acute Indigestion, Gorged Stomach, or Stomach Staggers of the Horse." I will quote freely, because it is the work of a man of great practical experience. He writes:

> Acute indigestion is a condition in which the stomach becomes tensely distended with food, movement of the muscular coat is arrested, and fermentation occurs in the food. Derangement of the nervous system, and semi-paralysis of the muscles of locomotion, especially those of the hindquarters supervene, followed by delirium, coma, and death. This is the usual course in the ordinary cases that occur in the Colony, but when an acute attack occurs in a healthy horse as a result of an excessive feed of grain, forage, etc., the symptoms develop suddenly, and closely resemble those of colic, and a fatal termination may occur from rupture of the stomach before any symptoms of brain disturbance appear.

He gives as the causes of "acute indigestion" (*a*) hurried ingestion of excessive quantity of food which is liable to ferment in the stomach, (*b*) enfeebled condition of the animal arising from exhaustion and long abstinence from food at the time when the full meal is eaten, (*c*) severe exertion immediately after a full feed, especially when it consists principally of grain, (*d*) a weak condition of the stomach itself due to a long continuance of feeding on rough, bulky, innutritious food, and (*e*) parasitic tumours in the walls of the stomach, chronic diseases of the liver, or the debilitating effects of some previous disease.

Then he writes:

> With respect to (*d*), this disease is most prevalent on the sour grass-veld districts, where the vegetation is coarse and innutritious. In such situations the horse requires to keep his stomach constantly distended with the coarse food in order to obtain sufficient nourishment. The consequences are that the walls of the stomach become permanently stretched, thinned, and weakened. . . . The irritation caused by the coarse food may and does appear to increase the desire for food, while it weakens the power to digest it. I have frequently observed a horse when suffering from indigestion manifest an eager desire for food, and even drop to sleep leaning his head on the manger with his mouth full.

Hutcheon's description of the symptoms of the ordinary type of case closely resembles that of chronic lactational dyspepsia of cows. *Post mortem*, there is a greatly distended stomach containing a sour fermenting mass of food. Occasionally the stomach may be found ruptured. Then Hutcheon states:

> The liver in a large majority of cases is more or less abnormal in appearance. In some cases it is atrophied and presents a tough leathery consistency; its cut surface is smooth, and its lobular structure indistinct. In other cases the liver is more or less indurated, cirrhotic, and is anæmic.

I am not dealing with cirrhosis of the liver in this paper, but I would say that I have a large amount of material which I may use in a later paper, and all of which goes to show that calcium and/or phosphorus deficiency is a main cause of this condition in animals, and probably in some cases of the human disease. Under this deficiency, the epithelium of the liver, kidney, pancreas, thyreoid, and elsewhere, undergoes atrophy and destruction just as it does in the intestine, the bronchi, and the epidermis, of which last, laminitis is a conspicuous example. The detriment to the nervous system belongs to the same primary cause, but it must very soon introduce secondary complications due to functional inefficiency. The gastro-intestinal and hepatic changes would be in part brought about by primary deficiency, but it seems quite clear also that another part is consequent on the failure of nerve function.

Passing from that, let me point to Hutcheon's mention of what one will recognize once more (if one thinks with me) as instinctive polyphagia induced by calcium deficiency or calcium phosphate deficiency. Hutcheon looks on it as the cause of the gastric condition. My view does not put it as antecedent, but makes both dependent on a common primary deficiency. At the same time it must be admitted that polyphagia would operate to fill the stomach more quickly than would be the case in an animal with only a slight appetite. The grass from the sour veld would contain only a minimum amount of calcium or phosphorus. That is my reason for attributing the polyphagia to that deficiency.

### Tenesmus.

At the caudal end of the bowel the symptom that corresponds essentially to colic is tenesmus, or straining. The autonomic control of this part of the bowel, however, is not carried on through the vagus, which is of cranial origin, but derives from the spinal cord. Tenesmus is a marked feature in cattle suffering from liver disease, and the evidence points strongly to calcium and/or phosphorus deficiency as the cause of this tenesmus as well as of the liver disease, but there is not space here to go into it properly.

### Tetany of Unstriped Muscle.

One may add here a further speculative remark. Spasmophilia and tetany are conditions brought

about usually (though not exclusively) by states of low blood calcium. Ordinary tetany is a cramp of voluntary muscles. Can we get cramps in the unstriped muscle of the intestine from the same cause? If it occurs, it would be manifested as colic. Apparently, we do not get it in the muscular coat of arteries from that cause, or is acrodynia something of the sort, and is some such condition the reason why cases of calcium deficiency have been explained as "ergotism"? Such views of possibilities are in no way incompatible with what has been said about colic in relation to disordered nerve function.

### Some Gastro-intestinal Ailments in Man.

#### *Chronic Dyspepsia.*

I have pointed to various disturbances of the visceral nerve apparatus as likely to be induced by deficiency of calcium and/or phosphorus, or by deficiency of vitamin *D*. But of course this does not mean an exclusive relationship. Lead poisoning and Gilruth's forage poisoning cases show that very plainly, and there are certainly other causes.

The horse has received a good deal of attention, because the material available has made that animal a convenient subject. Have we been taught any lessons that may help us in the study of disease in mankind? There are very suggestive analogies between some of the features of deficiency disease, as seen in experimental animals, and some forms of chronic visceral disorder in man. These analogies thrust themselves very persistently into notice. The "pot-belly" of rickets has been already considered, and classed as evidence of a functional neurosis.

Clinicians speak of atony of the stomach, also of dilated stomach, tympanic distension, gastrectasis, impaired motility, fermentation in the stomach. Also of disturbances of secretion such as hypersecretion, hyperacidity, achylia. And again of gastrodynia, gastric hyperæsthesia, gastrospasm, gastric vertigo, and palpitation or disordered action of the heart from gastric causes. What are all these?

In some patients with atonic functional derangements of the stomach, particularly when there is distension, vertigo is a very distressing symptom. It seems to me that this is precisely homologous with the nervous symptoms exhibited by a horse suffering from an acutely distended stomach due to impaction or tympany. These symptoms in the horse are popularly described as "stomach staggers," or "sleepy staggers," a name which interprets itself.

I did not mention gastroptosis or visceroptosis with the above list. It is associated with relaxation of the suspension apparatus, but it is hard to recognize muscular action as an intrinsic ingredient, though it goes with muscular relaxation. Yet it is worthy of remark that the viscera affected—alimentary canal, kidneys, and liver—are all within the area of distribution of the vagus nerve. As regards the mechanism of gastric atony, it will be agreed that the motor function of the stomach is disturbed, and in many cases, it seems, the secretory also. In gastric atony there is shown a defect of contractility, with loss of effective propulsive power, and this must owe its immediate cause to a change either in the nerve centre, the nerve fibre, the muscle tissue, or in some degree to a combination of those causes. The nerve and nerve centre to be affected in such a case would be the vagus and its nucleus.

#### *Gall Bladder Disease.*

After the stomach come the duodenum, the small intestine, the caecum, the appendix, and the colon, and in connexion with the duodenum are the gall bladder and bile ducts. How far motor inefficiency of the biliary apparatus is causally connected with stasis and with cholelithiasis, one cannot say with certainty. One may conjecture with some probability that it is one of the factors. As in the stomach, so also in the biliary passages, it is the vagus nerve that maintains muscular tone. The mechanism that provokes vomiting in biliary pain is doubtless a reflex through afferent fibres that pass to the vagus centre from the bile ducts or gall-bladder.

#### *Appendicitis.*

We have little information as to the conditions of functional inadequacy, motor or secretory, that affect the small intestine in conjunction with atony of the stomach. As to the appendix, we may conjecture, as we have in the case of the gall bladder, that a loss of muscular tone is likely to be a factor in producing chronic disorders, for instance, in favouring the formation of stercoliths that may on occasion provoke more acute troubles. It will be seen that a view like this would make some conditions of the appendix parallel with cholelithiasis, and it would also make some cases of acute appendicitis parallel with acute infections of the gall bladder. Moreover, it provides in a logical and reasonable way, a means of explaining the rather frequent association of a chronically diseased appendix with a chronically diseased gall bladder. I do not pretend that it is a matter to dogmatize about, for it would be unreasonable to push the parallelism too far.

#### *Chronic Constipation. Intestinal Stasis.*

When we come to consider the caecum and colon, a subject of much present interest comes into view, namely, intestinal stasis. There is no doubt that it is very frequently concomitant with an atonic and dilated stomach. There is a strong school of medical opinion which regards intestinal stasis as the chief cause of an enormous amount of ill health, mental and physical. I have no intention of plunging into a discussion of the relationship of chronic constipation or "intestinal stasis" to other complaints. I am sure there is good foundation for much that is said, but there are extremists who would seem to regard the average patient as a septic focus (colonic or otherwise) surrounded by protoplasm.

But what causes the intestinal stasis itself? For present purposes there is no need to look for every possible cause. There can be no doubt that one very

potent cause of chronic constipation is the relative deficiency in modern civilized dietary of what corresponds to "roughage" in the dietary of herbivorous animals. There is not sufficient coarse material in the food. But putting that aside, there is evidence that animals suffering from malnutrition, whether it be simple inanition or a relative deficiency of vitamins, or of mineral elements, or of requisite amino-acids, become subject to obstinate constipation, though sometimes this is replaced by diarrhœa. Suppose a man or woman whose customary dietary is at the same time small in total quantity, devoid of coarse material, and oscillating above and below the border of safety in one or more of the other necessary elements that I have referred to. We might expect intestinal stasis to be a chronic trouble in such a patient. But I have described here just the condition of things that does occur in many dyspeptic people, and especially if they are victims of the type of medical advice that insists on cutting off one thing after another from the dietary, a practice well enough for a few days, but the very worst for long periods.

**Summary.**

While it was shown in a previous paper that polyneuritis and paralysis were caused by calcium and/or phosphorus deficiency in the dietary, or by deficiency of vitamin *D*, further evidence of the fact is reviewed in the present paper.

The disturbance of nerve function in this group is very wide, so wide as to be comparable in extent and parallel in its effects to what is seen in lead poisoning, although so different in its pathogenesis.

The effects of this form of nerve dystrophy on the locomotor apparatus were discussed in the previous paper. Evidence is now produced which shows that the same deficiency causes corresponding detrimental effects in internal organs. There may be fatal bulbar paralysis. The autonomic nerve control exerted through the vagus nerve is disordered and weakened, causing a variety of serious secondary results.

These nerve dystrophies involving the internal organs are prominently seen in domesticated animals, but the condition of *laryngismus stridulus* seen in rhachitic children seems to belong to the class.

The phenomenon known as "roaring," relatively common in race-horses, is assigned to the same dietary cause. It is not due to an hereditary predisposition, as has been supposed, but is due to the inheritance of a traditional system of feeding. It is suggested that periodic ophthalmia, also relatively common in race-horses, is due to the same cause.

The liability of the stall-fed horse to a condition called "impaction," wherein an atonic stomach becomes distended with food, and the further liability to tympanic distension caused by fermentation of retained gastric contents, is referred to disordered autonomic control arising from calcium deficiency. The same explanation is given for the relatively great predisposition of the horse to colic, a predisposition not seen in the donkey. Under similar conditions of food deficiency, ruminants exhibit more or less similar symptoms. In ruminants, tenesmus may be prominent.

A similar explanation applies to the great dilatation of the stomach that is observed in monkeys and other animals suffering from rickets. The dilatation is not confined to the stomach. The "pot-belly" so characteristic a feature in rickets, is a consequence of the intra-abdominal distension.

Since the gall bladder is within the autonomic control of the vagus nerve, it is reasonable to expect in it a loss of tone parallel with loss of tone in the stomach and intestine. The vermiform appendix is subject to the same parallelism, and it is suggested that this may be a reason for their tendency to be associated in chronic disease.

In human beings it is possible that many cases of gastric atony and dilatation, and of intestinal stasis, may be explained as originating in a food deficiency, not necessarily always a calcium deficiency.

Further evidence is incidentally produced which goes to confirm the statement made in an earlier paper that it is a common characteristic of animals suffering from calcium deficiency to exhibit (among other things) polyphagia, and that this is an expression of protective physiological instinct.

A short reference is made to cirrhosis of the liver in animals, connecting it with calcium or calcium phosphate deficiency.

**References.**

(1) C. E. Corlette: "Observations on Calcium Deficiency as a Cause of Certain Diseases of Animals and Man," THE MEDICAL JOURNAL OF AUSTRALIA, February 18, 1928, page 198.

(2) C. E. Corlette: "The Paroxysmal Hæmoglobinuria of Horses and Cattle and of Man," THE MEDICAL JOURNAL OF AUSTRALIA, May 3, 1924, page 429.

(3) H. Wehrbein: "Paralysis of Pigs," *Journal of the American Veterinary Medical Association*, 1916, Volume XLIX, page 238.

(4) E. B. Hart, W. S. Miller and E. V. McCollum: "Further Studies on the Nutritive Deficiencies of Wheat and Grain Mixtures, and the Pathological Conditions Produced in Swine by Their Use," *Journal of Biological Chemistry*, 1916, Volume XXV, page 239.

(5) J. Law: "Dyspnœa Laryngea. Roaring. Hemiplegia Laryngea," in his "Textbook of Veterinary Medicine," Second Edition, 1905, Volume I, page 146.

(6) F. Hutyra and J. Marek: "Paralysis of the Recurrent Nerve," in their "Special Pathology and Therapeutics of the Diseases of Domestic Animals." Translation edited by Mohler and Eichhorn, 1913, Volume II, page 729.

(7) F. Smith: "Manual of Veterinary Physiology," Fifth Edition, 1921.

(8) E. Mellanby: "Experimental Rickets," Medical Research Council, Special Report Series, Number 61, 1921.

(9) J. Bland Sutton: "Observations on Rickets, &c., in Wild Animals," *Journal of Anatomy and Physiology*, 1884, Volume XVIII, page 363.

(10) J. Bland Sutton: "On the Diseases of the Carnivorous Animals in the Society's Gardens," *Proceedings of the Zoological Society of London*, 1884, page 179.

(11) J. Bland Sutton: "Bone Disease in Monkeys," *Transactions of the Pathological Society of London*, 1884, Volume XXXV, page 468.

(12) J. Bland Sutton: "Bone Disease in Wild Animals," *Transactions of the Pathological Society of London*, 1884, Volume XXXV, page 472.

(13) Chalmers Watson: "The Influence of an Excessive Meat Diet on Growth and Nutrition," *The Lancet*, 1906,

Volume II, page 1721. By reprint in Chalmers Watson's book, "Food and Feeding," 1913.
(14) R. McCarrison: "Studies in Deficiency Disease," 1921.
(15) D. Hutcheon: "Osteoporosis," *Agricultural Journal of the Cape of Good Hope*, 1905, Volume XXVI, page 540.
(16) J. Law: "Recurrent Ophthalmia in Solipeds. Periodic Ophthalmia. Moonblindness." In his "Textbook of Veterinary Medicine," Second Edition, 1906, Volume III, page 406.
(17) H. Ingle: "Osteoporosis in Animals," *Journal of Comparative Pathology and Therapeutics*," 1907, Volume XX, page 35.
(18) E. B. Hart, H. Steenbock and G. C. Humphrey: "Influence of Rations Restricted to the Oat Plant on Reproduction in Cattle," Agricultural Experiment Station of the University of Wisconsin, 1920, Research Bulletin 49.
(19) J. A. Gilruth: "Meningo-Encephalitis (Stomach Staggers) in Horses and Sheep," Fourteenth Report of the Department of Agriculture, New Zealand, 1906, page 293.
(20) J. A. Gilruth: "Paralysis in Horses and Cows due to Ingestion of Fodder," *Proceedings of the Royal Society of Victoria*, Volume XXV, New Series, Part 2, 1912, page 201.
(21) E. W. Hoare: "A System of Veterinary Medicine," 1915, Volume II, page 96.
(22) D. Hutcheon: "Acute Indigestion, Gorged Stomach, or Stomach Staggers of the Horse," *Agricultural Journal of the Cape of Good Hope*, 1899, Volume XV, page 588.

## ADVANCED PULMONARY TUBERCULOSIS TREATED BY THORACOPLASTY.

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IT is only about fifteen years since Wilms and Sauerbruch commenced performing paravertebral thoracoplasty on patients in whom it was not possible to induce a satisfactory artificial pneumothorax. During recent years the indications for this operation have been extended. When a lung cavity is situated very near the pleural surface, there are often adhesions extending to the thoracic wall. Each time a refill is given, these adhesions are dragged on and there is often a rise of temperature and there is a risk of hæmorrhage into the cavity or of tearing the cavity wall leading to a communication into the pneumothorax. It is more than probable that this is what happened in the sixth patient who within a few days of a refill rapidly developed a large purulent pleural effusion. Many are of the opinion that these cases of superficial cavity are more suitable for thoracoplastic than for artificial pneumothorax treatment. Another indication is the presence of a chronic empyema, whether tuberculous or due to mixed infection. The other indication illustrated in this series is gross displacement of the mediastinum and its contents through contraction of intrapulmonary fibrous tissue.

In this series of cases thoracoplasty was tried when artificial pneumothorax was impossible or had failed. All the patients had had many months of sanatorium treatment. Some did not have any free pleural space, while for those in whom it was possible to induce even a partial pneumothorax, this line of treatment was not abandoned until it was seen that it was impossible to induce the cavities to collapse by this method.

The permanent collapse of a lung is a serious operation, but artificial pneumothorax is not without its dangers and its complications. Pleural shock and air embolism are the two great dangers. Recurring pleural effusion, either serous or purulent, is a common complication, but the most serious ones are liable to arise when the pneumothorax is being absorbed. A considerable amount of fibrosis takes place within the tissues of the collapsed lung. When this is unsupported by the pressure of an artificial pneumothorax and the lung tissue commences to expand, there is great danger that cavity walls will be torn, producing extensive hæmorrhages, or the mediastinum and its contents will be dragged far over into the affected side. Both these complications can be minimized by a preliminary exeresis of the phrenic nerve, but they do not arise when a paravertebral thoracoplasty is performed.

If disease appear in the contralateral lung or if the extent of involvement of the contralateral lung increase, then artificial pneumothorax treatment can be stopped and the collapsed lung allowed to expand. It is then possible to consider the advisability of collapsing the lung in which the disease has recently appeared or extended. Thoracoplasty is, therefore, performed in those cases in which the chance of progression of any tuberculous lesion that may be present in the contralateral lung is relatively slight. It is usual to perform a preliminary exeresis of the phrenic nerve to test the sound lung. This was done in one patient, but not in the other in whom there was obvious involvement of the contralateral lung. This preliminary operation means a longer stay in hospital and an extra anæsthesia. In the second patient, in order to save extra expense, the operation on the phrenic nerve was performed at the same time as the first stage of the thoracoplasty.

Two of the patients were operated on in the Adelaide General Hospital. The remaining five operations were performed in private hospitals. This was made possible through the consideration of the anæsthetists, Dr. Gilbert Brown and Dr. Terrill. The economic problem was a very acute one for all these patients and it was because their condition was desperate in every way that such a radical procedure was advised, but the mortality from the operation of thoracoplasty is no greater than from that of artificial pneumothorax.

Exeresis of the phrenic nerve is usually done partly to ascertain whether the sound lung will tolerate the extra strain that is put upon it and also to test the individual. If his general condition as the result of this procedure deteriorates, it would be useless to go on with the more extensive operation.

By paralysing one half of the diaphragm and allowing it to ascend up into the thorax, the

capacity of that half of the thoracic cavity is lessened by one-third.

In the majority of the patients subjected to thoracoplasty this operation on the phrenic nerve was performed at the same time as the first stage. By aiding collapse of the lower lobe of the lung it was felt that it would lessen the possibility of an aspiration broncho-pneumonia of the lower lobe. Also the patients find it easier to cough, for they can use their abdominal muscles to cough with and so do not have to put so much strain on the recently divided intercostal muscles. It is very important that the patient should be able to start expelling the secretions of his lung as soon as he regains consciousness.

Five of the patients had cavities in one lung and pleural adhesions so that it was not possible to induce a satisfactory pneumothorax. The second and fifth patients had some involvement of the contralateral lung, but without this treatment the outlook was so hopeless that it was considered justifiable to take the risk.

CASE I. Mrs. T.H. had been out from England three years. Two years before coming to Australia she had pleurisy. She presented herself for examination on February 25, 1928. Eight months previously she had had a cold which had not compelled her to go to bed, but which had not cleared up satisfactorily; she had had some cough and sputum ever since. During the whole of this period her voice had been husky. Three months ago she was knocked down in a street accident and had spat up some blood. It was this which made her seek medical advice and tubercle bacilli were found in her sputum. She was sent to a sanatorium and from there to a private hospital where five attempts were made to induce an artificial pneumothorax. At the time of examination she had a temperature of 37·8° C. (100° F.) and a pulse rate of 92. There were signs of cavitation below the middle of the right clavicle. As all the previous attempts had been made in the right axilla, a spot just inside the angle of the right scapula was chosen for the pneumothorax. Four hundred cubic centimetres of air were introduced.

On March 3, 1928, the expiratory and inspiratory pressures were –0 and +6; after 800 cubic centimetres of air had been introduced the pressures were +4 and +10. There was no collapse of the right upper lobe.

On April 27, 1928, the pressures were –6 and +2; after a refill with 300 cubic centimetres they rose to +4 and +12. There was less cough and less sputum.

On June 5, 1928, there was blood in the sputum and no sign of collapse of the upper lobe.

For three months she had been having tuberculin injections and the size of the artificial pneumothorax became progressively smaller. When she was examined with the fluorescent screen it was seen that there was no collapse of the cavity; it was therefore decided to do a thoracoplasty. On June 9, 1928, the first stage was performed. Nine weeks after this she was screened again, when it was seen that there was good collapse of the right half of the thorax, the ribs were very oblique and there was no sign of the cavity. Her present condition is that her weight has increased by four kilograms (nine and a half pounds) in weight and she has no cough and no sputum.

CASE II. Mr. W.A. was aged thirty-nine years. Pulmonary tuberculosis was first diagnosed in 1924, when he was sent to a sanatorium for four and a half months and then he returned to work for three years. He appeared to be well till seven months ago, when he had a slight hæmoptysis and he returned to the sanatorium where two unsuccessful attempts were made to induce an artificial pneumothorax. On examination it was found that there was a cavity in the left upper lobe below the middle of the clavicle with involvement of the upper half of this lobe and signs of thickened pleura over the lower lobe. This was confirmed by means of an X ray photograph. Three days later he had a severe hæmoptysis for which his local doctor injected morphine. Later in the day he was put on to a mixture containing potassium bromide and sodium chloride. Within seven days there was pneumonic consolidation of the whole of the left lower lobe. His temperature was 40° C. (104° F.), his pulse rate 136 and his respirations 28. After four weeks the lower lobe showed signs of breaking down, being full of large bubbling *râles*. An effort was made to induce an artificial pneumothorax and it was found that there was no free pleural space in the axilla, but it was possible to introduce air near the angle of the left scapula. The expiratory and inspiratory pressures were –8 and +4; after 300 cubic centimetres of air had been introduced they were +4 and +7.

Many attempts were made to produce a satisfactory collapse of the lower lobe, but in spite of a positive pressure of 16 there was only a very limited pneumothorax. His cough was now very troublesome and his expectoration profuse. There were some fine moist sounds over the middle lobe of his right lung. As it was found impossible to increase the extent of his pneumothorax and to induce the lower lobe and the cavity in the upper lobe to collapse, it was decided to perform a thoracoplasty after a preliminary exeresis of the phrenic nerve. This preliminary operation did not affect his general condition nor did it appear to make any difference to the level of the diaphragm. Most probably this was because there was considerable thickening of the pleura over the lower lobe. His condition at the time of writing is that he is up and about, he has very little cough and sputum, he is gaining weight and his temperature does not exceed 37·2° C. (99° F.).

CASE III. Mrs. S., aged thirty-five, had had several months' treatment in a sanatorium where she had been treated with a right artificial pneumothorax. This was discontinued because of a purulent effusion. When first seen on February 27, 1928, her chief complaint was shortness of breath with some cough and sputum. The effusion was found to extend to within two and a half centimetres (one inch) of the lower border of the clavicle. On several occasions the effusion was aspirated and replaced by air, but when a screen examination was carried out, it was found that, although the upper and middle lobes were well collapsed, the lower lobe was almost fully expanded. To collapse the lower lobe it was necessary to induce a separate pneumothorax through a spot just internal to the angle of the scapula. By alternating these procedures, aspirating and replacing with air one week and refilling with air in the lower cavity the next week, it was possible to obtain a satisfactory collapse of the whole lung. On March 31, 1928, fifty cubic centimetres of air introduced into the upper cavity produced a positive pressure of 24 and on April 28, 1928, after aspiration of 230 cubic centimetres of purulent fluid, 150 cubic centimetres of air produced a positive pressure of 20 and in the lower sac, starting with pressures of –14 and –6, the introduction of seventy-five cubic centimetres of air raised the pressures to +6 and +10. As the purulent effusion recurred and the cough and sputum did not clear up, it was decided to perform a thoracoplasty and to avulse the right phrenic nerve at the first operation. After this was done she made an uninterrupted recovery and when her chest was screened at the end of September, the right thoracic wall was seen to be very well collapsed and there was no return of the effusion. She is now putting on weight, has very little cough and sputum and has a normal temperature.

CASE IV. Mrs. W.W., *ætatis* forty years, had a history of pulmonary tuberculosis for six years. She seemed to have recovered from her first attack until a year ago, when she developed a troublesome cough which produced a large amount of sputum; she lost weight and was very short of breath. Every evening her temperature rose to over 38·3° C. (101° F.). She was found to have a large cavity in the left upper lobe just below the middle of the clavicle and extending outwards towards the pleural surface. There was considerable infiltration of the remainder of the tissue of this lobe. Her fingers were clubbed and her finger nails were cyanosed. An attempt was made to induce an artificial pneumothorax through the upper part

of the left axilla, but the needle evidently entered the cavity for she immediately had quite a large hæmorrhage. After waiting for a week another attempt was made this time near the angle of the scapula and 300 cubic centimetres of air produced pressures of +10 and +16. Injections of tuberculin were given during the succeeding six months and at the end of that period the fluorescent screen showed that there was a long narrow pneumothorax extending from the diaphragm to where the pleura was adherent over the cavity, but that there was no pressure exerted on the cavity walls. She had much less cough than at the commencement of treatment and was not disturbed during the night. Every morning she expectorated a large amount of sputum and large bubbling *râles* appeared in the lung tissue below the cavity. As the attempts to induce the upper lobe to collapse had not been successful, it was decided to operate on the chest wall. Since the operation the cough and sputum have lessened considerably, she has gained weight and her temperature seldom goes above 37·2° C. (99° F.).

CASE V. Miss D.H. was aged twenty-eight years. After ten months in a sanatorium her condition had deteriorated and she was compelled to remain in bed, as she had a very troublesome cough, a large amount of sputum, her temperature went up over 37·8° C. (100° F.) every evening and she was progressively losing weight. Two attempts had been made to induce an artificial pneumothorax, but no free pleural space had been found. Examination showed that there was involvement of the left lung from apex to base with signs of cavitation in the upper part of the lower lobe and there was evidence of disease in the lower part of the upper and the middle lobes of the right lung. Her vocal cords were congested, but not ulcerated. She was put into hospital and attempts to induce an artificial pneumothorax were made in the axilla near the angle of the left scapula and below the middle of the clavicle, but it was impossible to find any free pleural space. It was therefore decided to perform a left thoracoplasty. The first stage and exeresis of the phrenic nerve were performed on September 15, 1928, and a fortnight after this an X ray photograph was taken. This showed ribs one to six still intact, the divided ends of the seventh and eighth ribs and cavities scattered throughout the substance of the left lung. After another week the operation was completed and after each stage she vomited for some days. While in hospital the amount of sputum was reduced from thirty cubic centimetres in twenty-four hours to less than four cubic centimetres. After leaving hospital she complained of pain in her left arm and then in her left hand, but this has gradually improved. Her pulse rate contiues to be high and is seldom below 100, her temperature is much lower than it was before the operation and although her sputum is very much less than it was, she is still troubled by a dry cough.

The five patients whose clinical histories are described, were submitted to the operation of thoracoplasty because they had cavities which could not be made to collapse by artificial pneumothorax. The third patient had an interlobar empyema which was tuberculous in nature and which kept recurring. It was necessary to perform this operation in the sixth patient, because he had a chronic empyema due to streptococci. Under a local anæsthesia the phrenic nerve was avulsed several weeks before the pleural effusion was drained. It was hoped that the diaphragm would ascend into the chest and so diminish the size of the pleural sac, but this did not happen. When fluid has been present in the pleural sac for some weeks, the diaphragm tends to settle in a low position and the pleura in contact with the effusion becomes very much thickened. Both these conditions were present, and prevented the success of this preliminary operation. In both patients who had a chronic empyema, the phrenic nerve was removed by Felix's phrenicus exeresis instead of by Gœtze's radical phrenicotomy in spite of the warning that the former might lead to the formation of a cold abscess or by infecting the mediastinum might lead to a fatal mediastinitis.

CASE VI. Mr. I., aged thirty-five, developed a *fistula in ano* in 1927 after twelve months' history of cough and expectoration. The anal condition was operated on by Dr. I. B. Jose who found a cavity in his right upper lobe and referred him for artificial pneumothorax treatment. This was commenced in January, 1928, and he started to improve at once, his cough and sputum lessened, he gained weight and his evening temperatures were lower. After his lung had been collapsed for three months and his temperature had become almost steady, he was given tuberculin injections. The complete collapse of his lung was prevented by an adhesion opposite the fissure between the upper and middle lobes. He never quite lost his cough and sputum, but was able to walk two miles a day and his temperature remained normal.

On July 8, 1928, he suddenly became very short of breath and when he tried to drink he found it difficult to swallow. On July 10 he was still very breathless with the same difficulty in swallowing and the trachea was seen to be displaced far over to the left. Eight hundred and fifty cubic centimetres of purulent fluid were withdrawn and the aspirations were continued for many weeks. On August 16, 1928, 750 cubic centimetres of blood stained fluid were withdrawn and some weeks later when a bacteriological examination was made of the fluid withdrawn, it yielded a pure culture of streptococci. During the first aspirations the fluid was replaced by air, but later the pleural sac was washed out with a weak iodine solution. Later still, after aspirating the fluid and washing out with an iodine solution, forty cubic centimetres of a 1% iodine solution were left in. The aspirating needle contaminated the tissues it passed through and he developed a lumbar abscess which first of all was repeatedly aspirated and then had to be drained. When the streptococci were found in the fluid, it was decided to do a thoracoplasty. There was a gap of over six weeks between the two stages and some union of the lower ribs had taken place and had to be broken down. For three weeks his temperature has not risen above 37·2° C. (99° F.), he has put on weight and there is very little discharge from the wound.

The third indication for thoracoplasty illustrated in this series is displacement of the mediastinum and its contents by the contraction of intrapulmonary fibrous tissue.

CASE VII. Mr. W., aged about forty years, had been in a sanatorium for two years. He still had some cough and sputum and was very short of breath. He complained that for some months he was seized every afternoon with severe pain in the right side of his thorax and it was so severe that he needed an injection of morphine and he had been advised to have his intercostal nerves injected with alcohol. On examination it was found that his heart was beating in the region of his right nipple and that the percussion note over the lower half of the right side of the thorax was quite dull. On exploring this dull area with a needle the pleura was found to be very thick and a small amount of purulent fluid was withdrawn. X ray examination revealed that the heart and mediastinum were dragged a long way over to the right. The left lung appeared to be quite healthy. He was advised to have thoracoplasty performed in order to give the mediastinum and its contents a chance to return nearer to the middle line. It was hoped that this would lessen his attacks of pain, increase the efficiency of his heart and aid the healing process in his lung. The second stage of this operation was performed less than two weeks ago and he has not yet lost his attacks of pain.

### Summary.

This series is composed of seven patients suffering from pulmonary tuberculosis on whom a Wilms-Sauerbruch thoracoplasty was performed and six of them had an exeresis of the phrenic nerve as well.

Six had unilateral cavities, but owing to pleural adhesions it was not possible to induce a satisfactory artificial pneumothorax. In addition two had chronic empyemata.

One patient had considerable displacement of the mediastinum caused by contraction of intrapulmonary fibrous tissue. He suffered severe attacks of pain and the relief of this was the main object of the operation.

Four of the patients are up and walking about and one of these is quite sputum-free; the others have much less cough and sputum. One is getting up for half the day, while the other two are still in bed convalescing after the operation.

It is as yet far too early to consider the question of cure in any of these patients, but in all of them the downhill course has been arrested and already five of them are definitely improved.

### Operative Technique.

The operation performed in all cases was an extrapleural paravertebral thoracoplasty of the Wilms-Sauerbruch type, with the addition of exeresis of the phrenic nerve, as recommended by Alexander. In all a two-stage operation was performed, the stages being separated by a period varying from two to six weeks, the length of the interval being determined by the general condition of the patient and the local wound condition. The longest interval was six weeks and by that time there was already considerable regeneration of the ribs and a longer interval would certainly tend to prevent the achievement of a complete collapse of the chest wall.

Anæsthesia was obtained by nitrous oxide-oxygen or ethylene-oxygen inhalation in all patients but one for whom gas was not available, and a second stage operation (Case VI) was performed under ether. No ill effect has been noticed since this administration of ether, but undoubtedly gas or ethylene is more suitable in every way. Local anæsthesia is favoured on the Continent and renders it possible to have the patient sitting forward during the whole operation, thereby facilitating displacement of the scapula and giving better exposure to the upper ribs. On the other hand, local anæsthesia has its defects. Collapse may follow the use of large quantities of "Novocain," even in dilute solutions and the combined apprehension and shock of the operation throw a big strain on a patient already seriously ill and toxic.

The only disadvantage of gas or ethylene and oxygen is that of obtaining a position which permits the administration of the anæsthetic without limiting the exposure of the upper ribs. With the assistance of Dr. Gilbert Brown the following position was devised and served the purpose. The patient was anæsthetized lying on his back and the phrenic nerve was then slowly but completely avulsed; the avulsion of twenty or more centimetres made it certain that any accessory phrenic fibres had been interrupted. The patient was then turned on to his face, his affected lung being kept undermost while he was being turned to avoid emptying the contents of a cavity into his bronchi or trachea. Before the administration of the anæsthetic was begun, the patient was asked to empty his cavity himself as far as possible.

He was then so placed that the affected side of his chest was almost over the side of the table, with the arm of that side hanging freely down; the wrist was loosely slung by a clove hitch to the pillow. This position allowed the scapula to fall away from the thorax when the scapulo-vertebral muscles were divided. The head was turned away from the side of the operation and the head-piece of the table dropped; this lessened the difficulties of the anæsthetist and opened out the region between the scapula and the cervical vertebræ.

The remainder of the first stage operation consisted of the resection of from ten to twelve and a half centimetres (four to five inches) of the fifth to the eleventh ribs inclusive, through a long J-shaped incision; the ribs are resected as far back towards the transverse processes of the vertebræ as possible. Hæmorrhage is free from vessels in the post-vertebral muscles, but soon ceases. The scapulo-vertebral muscles are accurately sutured, the wound is drained (for thirty-six hours) by a long roll of rubber dam which is brought out of the bottom of the incision.

The second stage was performed through a similar, but higher incision with the patient in the same position. An endeavour was made to remove one and a quarter centimetres (half an inch) of the first rib and as much as possible of the remaining three ribs, usually about five to seven and a half centimetres of each. It was found to be easiest to strip the first rib before the others, but to leave its actual division to the end. It was divided by bone forceps. The wound was closed as before. Division of the first rib is essential; the more one was able to remove, the better the collapse.

In patients with great pleural involvement it was astonishing to note the degree of hypertrophy of the ribs. Often they were found to be quite triangular in section, with the apex directed inwards, instead of presenting the usual flattened quadrilateral section.

In only two patients was avulsion of the phrenic nerve done as a preliminary operation; in all the others it was combined with the first stage operation. From the published results it appears to be well worth doing, particularly as it lessens the risk of a subsequent aspiration pneumonia in the lower lobe. The operation of phrenic avulsion is not without risk. The position of the nerve is fairly constant upon the anterior surface of the *scalenus anticus*, passing downwards and inwards. A large supraclavicular branch of the cervical plexus is often encountered, but is passing down and outwards and is superficial. A venous plexus is often encountered, also the thoracic duct network, if the incision be too low. The essential point is to expose the *scalenus anticus* fibres on which the nerve will be found after its branches have pierced

that muscle. The nerve is divided and slowly twisted out; unless done gently there is a risk of mediastinal hæmorrhage and of mediastinal infection in cases of tubercular empyema.

#### Post-operative Treatment.

The patient is allowed to sit up as soon as he returns to his bed and nourishing fluids are given forthwith. There is always considerable intercostal pain for a few days and for this morphine is given in doses insufficient to prevent coughing. The immediate effect of the collapse is an increase of sputum; it is essential that the patient should be able to expectorate freely. A firm binder gives support to the collapsed chest wall and assists the effort of coughing. The rubber drain is removed in thirty-six hours; there was generally a large amount of serous and bloody oozing, but most of the wounds were completely healed in two weeks. After the second stage operation, the drop of the shoulder girdle caused traction on the brachial plexus and the pain produced was relieved by maintaining a firm support under the point of the elbow and so raising the shoulder.

## THE SAFETY OF ETHYL CHLORIDE AND THE POSITION OF A PATIENT DURING INDUCTION AND ADMINISTRATION OF A GENERAL ANÆSTHETIC.

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AT a coroner's inquiry into a death under ethyl chloride in Melbourne, the question was raised as to the safety of ethyl chloride as an anæsthetic.

Like every other anæsthetic, there is a risk in its administration under certain conditions, especially by the closed method, that is a method in which air is not admitted to the patient during induction, but given a free supply of air during induction and used in either the open or semi-open method, it is one of the safest of all anæsthetics, not even excepting nitrous oxide and oxygen. The above remarks do not mean that you can be free and easy with it; no anæsthetist at any time can ever be free and easy with any anæsthetic.

By the semi-open method the air valve in the inhaler which should have a diameter of not less than twelve and a half millimetres (half an inch), is gradually closed over as induction proceeds, so that by the time induction is completed which is as a rule in from one and a half to two and a half minutes, the air valve is fully closed. This will give from three to five minutes full anæsthesia.

Ethyl chloride is not a drug that should be used for prolonged anæsthesia, nor is it intended that it should so be used, except under very exceptional circumstances and in the hands of an expert. This is for two very valid reasons, its high and I think quite unnecessarily high cost and also its wastefulness when used for any length of time by the open method.

It is one of the very best of all the anæsthetics that we have for short operations of from one to five minutes or for preliminary induction to ether. In using the words ethyl chloride I include also "Somnoform" which is 83% chloride of ethyl, 16% bromide of ethyl and 1% methyl bromide.

At the Melbourne Dental Hospital and the Melbourne Eye and Ear Hospital it has been used in considerably over 75,000 patients of all ages during the past twenty-three years and there have been two deaths, one of which, I think, should not have occurred. The collapse occurred during a second administration of ethyl chloride to an exceptionally delicate female patient who had recently been through a very severe hæmorrhage and was not in my opinion a fit subject for the second administration of an anæsthetic, either local or general.

This morning, December 6, on reading the report of the inquest in the morning papers, I decided to test for myself the so-called dangerous effects of ethyl chloride under what would normally be considered disadvantageous circumstances. Feeling a bit "off colour" with a threatening cold, I took one gramme (fifteen grains) of antipyrine and 0·3 gramme (five grains) of citrate of caffeine in half a glass of hot water. This was followed by a good breakfast. I then went down to the Melbourne Dental Hospital and had the following administrations given to me by Dr. Aird.

These administrations were all given sitting upright in the dental chair.

No. 1—

Pulse before induction, 100, good, regular.

Blood pressure before induction, 158 millimetres of mercury.

Anæsthetic, ethyl chloride, eight cubic centimetres by the semi-open method.

Pulse one minute after induction began, 104.

Blood pressure one minute after commencing induction, 170 millimetres.

Pulse two minutes after commencing induction, 102.

Blood pressure two minutes after commencing induction, 162 millimetres.

Time of induction, two minutes ten seconds.

Duration of anæsthesia, five and a half minutes.

No. 2.—There was then an interval of fifteen minutes before the second administration, this time of "Somnoform" by the semi-open method.

Pulse before commencing administration, 80.

Blood pressure before commencing administration, 158 millimetres.

Amount of "Somnoform" used, five cubic centimetres.

Pulse after one minute administration, 104.

Blood pressure after one minute administration, 176 millimetres.

Pulse after two minutes administration, 104.

Blood pressure after two minutes administration, 164 millimetres.

Time taken to induce full anæsthesia, two minutes 35 seconds.

Duration of anæsthesia, four and a half minutes.

No. 3.—There was a further interval of fifteen minutes; this time the ethyl chloride was administered by the open method.

Pulse before commencing administration, 84.

Blood pressure before induction commenced, 156 millimetres.

Amount of ethyl chloride used, 13 cubic centimetres.

Time taken in inducing full anæsthesia, two and a quarter minutes.

Pulse after one minute of induction, 102.

Blood pressure after one minute of induction, 150 millimetres.

Pulse after two minutes of induction, 104.

Blood pressure after two minutes of induction, 156 millimetres.

Duration of anæsthesia, five minutes forty seconds.

The first administration was commenced at 11.35 and the third at 12.20 p.m.

Following upon the third administration, I felt slightly sick, but with no real tendency to vomit. I left the hospital at 12.45. After a cocktail as a pick-me-up I enjoyed a hearty lunch at 1 p.m.

It will be noticed that blood pressure rose during induction by the semi-open method on both occasions and remained about normal during induction by the open method. The pulse rate also rose during induction and was always of good volume.

In every instance the anæsthetic was taken quietly without any attempt on the part of the patient to struggle and in all three administrations the induction was of a quite pleasant nature and at no time was there the least feeling of want of air, fullness in the head or pulsation of the heart, but just a very pleasant sensation of drifting off to sleep, with a creepy feeling gradually spreading from the feet upwards.

At the Melbourne Dental Hospital all general anæsthetics are given in the upright position at an angle of about eighty degrees.

In my own work for all classes of cases and patients of all ages I always prefer the head and shoulders well raised and at an angle comfortable for the patient during induction, no matter what general anæsthetic is used.

This comfort of the patient during induction is an essential. No patient can take an anæsthetic well if he feels that he is in an uncomfortable position whilst he is going under. The same patient who will resist going under in a head low or supine position, will take chloroform quite naturally and quietly if sitting comfortably in an arm chair. He will have none of that feeling of fullness in the head or increased pumping action of the heart so frequent and so very disturbing to a full-blooded patient when placed in the supine position. The ease and comfort with which a patient will take a general anæsthetic in the upright position as against the distress felt by the patient when in the supine position was illustrated on several occasions when I was able to give chloroform and ether to able-bodied men in the Royal Navy and Royal Australian Navy during the war. I was fortunate in being able to try both the supine and the upright positions during induction on the same patient in many instances and the lurid remarks passed by these men against the supine position as compared with the ease and comfort of the upright or semi-upright during induction were very instructive.

In all general anæsthetic work there is during induction a tendency to cerebral congestion, which means cerebral excitement and a tendency to struggle; this is especially the case if the patient is not comfortable. No anæsthetist should commence his work until he is assured of the comfort of his patient.

Not only is there less tendency to struggle, which means there is less strain thrown on a heart which may be abnormal, but also there is less anæsthetic required to place the patient in an anæsthetic stage. We are working with the advantage of a not over-congested brain.

The ease and comfort of a patient during induction means safety. The anæsthetist can always lower the patient quietly to a more supine position for the comfort of the surgeon after induction, should it be necessary to do so.

Personally, I always prefer to retain, whenever possible and not inconvenient to the surgeon, a certain amount of head and shoulder elevation throughout the operation.

After over thirty years of anæsthetic work in all classes of patients and of all ages, without an anæsthetic fatality, I do not think it can be entirely due to luck.

I have never been able to see the common sense of the head low position. We do not want hyperæmic brains, at least not during induction; what we require is a brain as anæmic as possible; we can get all the congestion required from the anæsthetic itself during induction.

A not over-congested brain means a quiet, even induction; a congested brain means struggling, full feeling in the head, very often accompanied by noises and a general uncomfortable sensation with an extra strain thrown on the heart. If that heart happens to be abnormal or lacking in tone, then possible collapse and death may occur which might have been avoided.

## Reports of Cases.

### CEREBRAL TUMOUR WITH UNCINATE FITS.

By W. WILSON INGRAM, M.C., M.D. (Aberdeen),
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AND
S. V. MARSHALL, M.B. (Sydney),
*Medical Superintendent, Royal North Shore Hospital, Sydney.*

#### Clinical History.

L.T., A MALE, aged thirty-seven years, was referred to the Hospital on October 5, 1928, for an opaque meal and

X ray examination of his gastro-intestinal tract. He complained of headaches of eight months' duration and of vomiting for six weeks. During the latter period he had had failing vision and for the two weeks preceding his admission he had lost completely the sight of his left eye.

His father had died of colitis. His mother, one brother and four sisters were stated to be in good health. One brother had died of blood poisoning and another in infancy from an unknown cause.

He was born and had lived all his life in New South Wales and he had led an active life as a labourer. He was a moderate smoker and drinker. He denied having suffered from any form of venereal disease, but stated that for some months he had been impotent.

Previous illnesses consisted of a left facial paralysis two years previously from which he recovered completely. Recently he had been in a local hospital for three weeks under observation for the vomiting.

The headaches were located chiefly in the occipital region and the back of the neck, but at times were present in the left frontal region. They were of a continuous aching character.

The vomiting was intermittent and would occur without provocation at any time of the day or night. The amounts were small and the colour of the vomitus yellowish. In addition he had noticed nasty tastes in his mouth and bad smells assailed him. His family noticed that he champed his teeth and smacked his lips a good deal and they were worried over the fact that he used to go about the house looking for gas leaks which did not exist. The patient complained that he had lost his former energy and that his mind seemed to be dull, while his friends often told him that he was becoming very "dopey."

Microphotograph × 400, showing elongated cells, multinucleated cells, areas of degeneration and capillaries (hæmatoxylin and eosin).

On examination he was seen to be a rather poorly nourished man approaching middle age. Considerable mental dulness was present, but intelligent replies were made to questions after some delay. His speech was slow, but not monotonous. His gait was steady and slow, but the equilibrium appeared to be good. A left-sided ptosis was a very noticeable feature.

Examination of the nervous system revealed some loss of power of the right upper extremity; the lower limbs were normal. The various tendon reflexes were normal, while the plantar reflex was flexor on both sides. There was no impairment of sensation. An examination of the *fundi oculorum* revealed an intense papillœdema, especially pronounced on the left side. Apart from this and the above mentioned ptosis no defect of the cranial nerves was observed. By means of lumbar puncture thirty cubic centimetres of clear cerebro-spinal fluid were obtained under definitely increased pressure. The Wassermann and Kahn's floculation tests on the cerebro-spinal fluid both yielded no reaction, while the cellular constituents were normal.

The temperature remained normal throughout, while the average pulse rate for the period of his stay in hospital was 74·7 per minute.

No abnormal constituents were detected in the urine and no abnormality was discovered in his other systems.

A diagnosis of cerebral tumour of the left temporal lobe involving the uncinate convolution was made.

He remained very drowsy and ten days after his admission to hospital he lapsed into a state of coma from which he could not be roused. Hypertonic saline solution was given in an attempt to reduce the intracranial pressure, but he died twenty-four hours later without regaining consciousness.

### Post Mortem Findings.

*Post mortem*, on opening the skull the meninges on the left side were found to be matted together and adherent to the temporal bone. The whole of the anterior portion of the left temporal lobe was involved in an area of softening and owing to this and to the adhesions the brain was removed with difficulty. From the centre of the degenerated area a spherical firm tumour 1·27 centimetres in diameter slipped out. It had a short pedicle at one side. The other organs of the body appeared to be healthy.

Microscopically the tumour was found to be extremely cellular. The cells were of varied sizes and shapes. Some were small oval or rounded. Others were elongated with an oval nucleus and two processes — the bipolar spongioblasts; they were the predominant cell. In addition numerous large multinucleated cells were scattered throughout the section. The growth was rich in capillaries and there were numerous areas of degeneration. Round the vessels and at the periphery were numerous small round cells and leucocytes. The primitive bipolar spongioblasts could be seen in apparent rosettes round the capillaries and in pallisade arrangement throughout the growth.

We are indebted to Dr. Oliver Latham, of the Department of Mental Hospitals, for showing us numerous beautiful preparations of cerebral tumours illustrating more primitive and higher types. With his help and with the aid of these we are convinced that the tumour falls into the group of *spongioblastoma multiforme*.

### Bibliography.

George Bell and Oliver Latham: "Tumour of the Brain," THE MEDICAL JOURNAL OF AUSTRALIA, December 15, 1928, page 757.

E. A. Carmichael: "Cerebral Gliomata," *Journal of Pathology and Bacteriology*, July, 1928, page 493.

# Reviews.

## THE PRINCIPLES OF PHYSICS.

PROFESSOR RUSS in his book "Physics for Medical Students" has succeeded in compressing a large amount of matter into a very small space, as may be gathered from the fact that the main principles of mechanics, heat, light, sound, electro-statics, magnetism, current electricity, electro-magnetism, X rays and radio-activity are discussed within the limits of 225 pages of good, large type.[1]

This feat appears the more remarkable when we find that not only are numerous diagrams interpolated throughout the text, but also several quantitative examples of the application of various formulæ.

The treatment naturally is condensed to a high degree, but is extremely well done, the volume furnishing a lucid and fascinating *résumé* for one already versed in natural philosophy. It is doubtful, however, whether the previously uninformed student could digest the subject matter in so concentrated a form, though the book should serve admirably as the basis for a course of lectures and the corresponding examination.

## RADIOLOGICAL EXAMINATIONS.

"RÖNTGENOLOGY: THE BORDERLANDS OF THE NORMAL AND EARLY PATHOLOGICAL IN THE SKIAGRAM," by Alban Köhler, embodies a description of the various phases of Röntgenology as observed by the author who has had a most extensive experience in this subject, and includes various views of sundry other workers in this field.[2] The special feature of the work is the voluminous bibliography.

The illustrations are very numerous, but most of them are diagrammatic only. This is rather unfortunate, although it has the merit of conserving space. In the earlier days of Röntgenology, when skiagrams were much less clear, especially in the absence of the Potter-Bucky diaphragms, these would have been fully justified, but more well chosen, clearly reproduced photographs would be far more convincing and would have a greater educational value than drawings in more recent times.

The translation, unfortunately, is not good, is frequently ungrammatical, and poorly chosen words and expressions have been selected to express the German ideas. In German, for instance, it is well understood what is meant by the first, second, third and fourth fingers, but much confusion is caused in English by the use of the same terms. Likewise, "lower arm" is not an anatomical term usual in the English language, but it would be pedantic to indicate numerous other shortcomings in this respect.

Many of the various anatomical and early pathological features are well described. To Köhler's disease of the second metatarsal bone eighteen pages are devoted and seven to Köhler's disease of the scaphoid, to which the author is responsible for first directing attention. Other lesions do not receive the same detailed description.

Those in search of particulars of intrathecal injection of "Lipiodol" or even the more important procedures of pyelography will be greatly disappointed with the statement: "As this book is primarily intended for practitioners, the description of pyelography findings will not be attempted." It is a great pity that the section dealing with the gall bladder was not entirely scrapped and revised in view of the voluminous work done since the studies of Cole and Graham. The latter as well as references to the bibliography are mentioned, but a systematic description of cholecystography has not even been attempted. On the other hand, the section devoted to the heart is elaborate and extends over forty-eight pages.

The book represents the findings of a keen observer with very considerable experience, together with the observations of a large number of other workers in the same field. Its arrangement of bibliography is most excellent. However, although the author considers that the practitioner does not need any description of such recent works as are written on pyelography and cholecystography, yet it must be inferred that he would be much more interested in the voluminous description, for instance, of the heart shadow.

# Notes on Books.

## NOTES ON SOME DIARIES AND OTHER BOOKS.

NOT long ago we received from the Manager of the Sydney branch of the great firm of Parke, Davis and Company a neat little book entitled "Lectures on Biological Products." This publication is in essence a clear, concise and instructive outline of ordinary bacteriological methods, a short *résumé* of the more important pathogenic bacteria and of the current views on immunity and a description of the laboratory preparation of antitoxins, serums, vaccines, biological extracts and the like. Much of the information is naturally contained in the text-books on bacteriology and immunity, but the presentation of the subjects is different and matter concerned with the preparation on a large scale of antitoxin and other therapeutic serum is not often encountered in books on bacteriology. The illustrations are good and well chosen. We understand that the edition of this little book, which was published in London in 1928, has been nearly exhausted. We congratulate the firm on an excellent little publication.

The German firm "Behringwerke" of Marburg on the Lahn has issued an artistic brochure descriptive of the methods of preparation of therapeutic serums, vaccines and other biological preparations. The matter is amplified by the inclusion of information concerning the use of the several preparations of this firm. The booklet is profusely illustrated and the reproductions are of a very high order. The description of the methods of preparation of serums and other products and of their uses is in the German language. The information is well given and is sufficiently complete to interest medical practitioners with some knowledge of bacteriology and immunology. The firm that was founded by von Behring and that bears his name, enjoys the reputation throughout Europe of producing reliable biological preparations. It would appear to us that the brochure will be found to be extremely useful to medical practitioners who read and speak German. It contains much of practical value and is reliable in its details.

The Anglo-French Drug Company, Limited, of Gray's Inn Road, London, has sent us some very useful diaries, in the form of pads or bunches of sheets, one for each day of the year with ample space for daily notes. Attached to the cover is a concise list of the pharmaceutical products of this firm. Each preparation is described, its composition is explained, directions for use are given and other essential information is attached. It covers fifty pages of matter. We commend this little publication to the medical profession. From the therapeutic point of view there is much worth learning in its pages.

Oppenheimer, Son and Company, Limited, of 179, Queen Victoria Street, London, have again sent us their "*Ephemeris Pharmacologica*." It is unnecessary to describe this diary, for it has become familiar to the medical profession in the course of many years. Many additions have been made; a great deal of the matter has been revised and the information as it now appears is extraordinarily complete and quite reliable. The chapters on poisons and their antidotes and on drugs and their doses serve as useful reminders to the busy practitioner. The therapeutic notes also have many uses. The diary part of the book is admirably suited to the requirements of those with a considerable visiting list. We congratulate the firm on maintaining a high standard for their diary.

[1] "Physics for Medical Students," by Sidney Russ, D.Sc. (London), F.Inst.P.; 1928. Edinburgh: E. and S. Livingstone. Demy 8vo., pp. 236. Price: 10s. 6d. net.

[2] "Röntgenology: The Borderlands of the Normal and Early Pathological in the Skiagram," by Alban Köhler, Prof. Dr. Med., Wiesbaden; rendered into English from the Fifth German Edition by Arthur Turnbull, M.A., B.Sc., M.B., Ch.B. (Glasg.). London: Baillière, Tindall and Cox. Crown 8vo., pp. 574, with illustrations. Price: 42s. net.

The Medical Journal of Australia

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## The Surgeon's Fees.

In a discussion on the splitting of fees that took place at the annual meeting of the College of Surgeons of Australasia on March 6, 1929, Dr. J. Lockhart Gibson remarked that since every fellow of the College signed a pledge that he would not divide fees, further consideration of the subject could not be permitted. In spite of this an extraordinary debate took place and one fellow of the College dared to ask his colleagues to sanction the practice to the extent of five guineas. The Victorian Branch of the British Medical Association has offered a complicated and cumbersome definition of division of fees. Unfortunately this definition narrows the subject to the paying of part of the fee received by one medical practitioner to another person as a consideration for the introduction of the patient. It is obvious that few, if any, medical practitioners would admit that a sum of money received from a surgeon or other practitioner was accepted as payment "for being influential or instrumental in bringing the patient to the specialist or practitioner for advice, operation or other treatment." If this definition were adopted, it would be impossible to find anyone guilty of the offence, save in very rare circumstances. If a definition of division of fees is actually needed, it should be simple and unrestricted. We should suggest that division of fees, fee splitting or dichotomy, whichever term be employed, be taken to mean the handing of a part of the fee received from the patient by one practitioner to another person for some service rendered or to be rendered in connexion with the patient. It must be remembered that one form of this illegal and objectionable practice is the payment by the specialist to the general medical practitioner of a substantial part of the fee, ostensibly as payment for assistance at an operation or examination, but actually as an inducement for the general practitioner to call in the specialist on other occasions. It is a proffered bribe, not demanded by the introducer and not "a transaction for financial gain practised under a contract understanding or by consent, silent or spoken." While it is gratifying to note that Dr. J. Newman Morris has been informed by counsel that if a practitioner were convicted of a misdemeanour under *The Crimes Act*, 1915, the maximum penalty would be imposed, it is doubtful whether in the circumstances set out a conviction could be established. Let it be supposed that a surgeon received fifty guineas from a patient for an operation and that he handed fifteen guineas to the general practitioner who called him in and who assisted him at the operation. It would be argued that a fee of fifteen guineas is not excessive for efficient assistance.

The fellows of the College of Surgeons of Australasia resolved that when a surgeon collects fees for the anæsthetist or for his assistant, the amount of the fees, with the names of the respective recipients, should be definitely stated on the account rendered to the patient or the patient's friends. The subject, however, is to be further considered. Is there really any difficulty? If the surgeon and the general practitioner intend to behave as honourable men, none will arise. It is a rule of the Royal College of Physicians of London that no member or fellow may sue for the recovery of fees. As a result consulting physicians have adopted the practice of collecting their fees at the time of the consultation and therefore have no need to render accounts. If a surgeon followed the same rule, his financial dealings with the patient would be limited to the reception of a cheque at the time when the service is rendered. The correct procedure would be that the general practitioner would inquire of the surgeon what his fee would be and what fees should be paid to the anæsthetist and the assistant. This information would be passed on to the patient who would be asked to pay the surgeon, the anæsthetist and the assistant individually. In the event that the general practitioner acts as assistant, he would be wise to inform the patient that his fee for assisting at the operation amounting to a given sum would be charged in his usual account.

If this method were adopted as the proper one, any attempt on the part of either the surgeon or

the general practitioner to divide the fee paid by the patient would necessarily amount to a secret commission. If both parties were dishonourable, it might be difficult to obtain information of the transaction, unless, indeed, one or other became indiscreet. Information concerning the willingness of a surgeon or a general practitioner to give or to accept a secret commission could be obtained when one or other made overtures with a view to the establishment of an understanding, or if a sum of money were actually offered to an unwilling practitioner. In such circumstances the maintenance of the honour of the medical profession would be endangered if the unwilling member did not report the matter to the Branch of the British Medical Association to which he is attached and to the Medical Board. Whether he should inform the patient or not would be a matter on which he should take legal advice. Dichotomy would become rare, if a few practitioners were convicted and sentenced to two years' imprisonment for having attempted to offer or to obtain a secret commission.

## Current Comment.

### LOBAR PNEUMONIA.

ACUTE lobar pneumonia is generally described as being due to an inflammation which starts at one place and spreads by direct continuity, involving various structures in its course and leading to extensive consolidation of the lung substance. Four stages in the process are described—acute congestion, red hepatization, grey hepatization and resolution. These stages have been recognized as a result of clinical observation and *post mortem* findings. There are several aspects of the condition which are not covered by these generalizations. One of the most important is that of the crisis and how it is brought about; this is obviously bound up with the immunological side of the question. There is also the question of the predilection of pneumonic infection for the lower lobes of the lungs and that of the method of infection and the way in which the lesion in the lung is initiated.

The subject has recently been discussed by Pol N. Coryllos and George L. Birnbaum at the annual meeting of the American Association for Thoracic Surgery.[1] They put forward an interesting theory and support it with some experimental work and clinical observations. They believe that lobar pneumonia must be regarded as the pneumonic variety in the large category of obstructive atelectasis of the lung. They believe that pneumonic infection of the bronchial tree is not by itself sufficient to produce lobar pneumonia. Another factor besides the presence of the pneumococcus is necessary and this is the occlusion of a bronchus by the pneumonic exudate. In the earliest stages lobar pneumonia presents radiographic and auscultatory signs of a lobar atelectasis, due to the obstruction of a bronchus; the signs appear to proceed from the periphery to the centre of the lobe, because the absorption of the alveolar air is more pronounced in the periphery where the alveoli are more numerous. Simultaneously a cellulitis spreads from the hilar portion of the lobe to the periphery, accompanied by congestion, interstitial and lymphatic infiltration, vascular engorgement and the production of the characteristic exudate. Thus, after the initial and transitory period of simple atelectasis there is a period of pneumococcic invasion. Coryllos and Birnbaum go on to state that crisis or lysis is due to the disintegration of the fibrinous exudate of grey hepatization and to the sudden liberation of a large bronchus, thus allowing a sudden drainage of the affected lobe. If the liberation occurs early, there is an abortive form of the disease or an early crisis. If the obstruction of the bronchus is prolonged, greater alterations of the bronchi and parenchyma of the lung will follow and in this way an explanation is afforded for postpneumonic abscess, bronchiectatic lesions *et cetera*. The localization of the pneumonic atelectasis is dependent on the same laws as those governing massive atelectasis. For purely mechanical reasons it is more frequent in the inferior lobes, particularly on the right side. The only distinction between massive and patchy atelectasis is a difference in the size of the obstructed bronchus. The same may be said of pneumococcal atelectasis; if a large bronchus is occluded, there will be lobar pneumonia; if several small bronchi are obstructed, bronchopneumonia will result. Coryllos and Birnbaum also lay stress on the part played by the ciliated epithelium. To failure of action of the cilia they attribute the rapid onset of pneumonia in drunkards, the decrease (in part) of resistance of the lung under the influence of cold and possibly the frequent recurrence of pneumonia in the same lobe.

In their experiments Coryllos and Birnbaum investigated the following problems: (i) the experimental production in dogs of pneumococcal "lobar pneumonia," (ii) the interpretation of the clinical and radiological signs in experimental lobar pneumonia with special reference to massive atelectasis, (iii) the influence of bronchial obstruction on the production and evolution of the disease after infecting the corresponding lobe with a culture of the pneumococcus, (iv) the influence of decreased oxygen tension on the development of pneumococcus cultures, (v) the effects of the liberation of a bronchus by the aspiration of the exudate contained within it. It is not proposed to give details of these experiments owing to limitation of space. Reference will be made to them when they have direct bearing on the points in discussion.

[1] *Archives of Surgery*, January, 1929, Part II.

After describing their experiments Coryllos and Birnbaum go on to quote the results obtained by others and conclude that the following points appear to be definitely proved: (i) The bronchogenous origin of pneumonia, (ii) the beginning of the lesion at the hilar portion of the lobe, (iii) the atelectatic appearance of the lobe from the start, (iv) the relation of the virulence of the pneumococcus to the amount of fibrin in the exudate, (v) the relation of the amount of fibrin to the evolution of the disease: the poorer in fibrin the exudate, the quicker the recovery is apt to be. The first point to which attention must be drawn, is that Coryllos and Birnbaum have made a fundamental error in applying without reserve to human beings the results of animal experiments on the method of production of pneumonia. They do not discuss the hæmatogenous origin of pneumonia. They point out that Wadsworth, Lamar, Meltzer, Blake and Cecil have experimentally proved the bronchogenous origin of pneumonia and that there is no definite experimental proof of the hæmatogenous theory. The experimental evidence does not by any means exclude an origin other than bronchogenous and it is fallacious to build up an elaborate theory of the pathogenesis of pneumonia on a view which is not proven. This must be kept in mind during the further consideration of the work of these two observers.

In considering the definition of Coryllos and Birnbaum of pneumonia as the pneumococcal variety of obstructive atelectasis, it must be remembered that there must necessarily be an infection of the bronchial walls before a pneumococcal exudate can be thrown out. The atelectasis is thus secondary to an infection of the lung. In other words the statement that after the initial and transitory period of simple atelectasis there is a period of pneumococcic invasion is pure hypothesis. When once the bronchus has been blocked, it is quite conceivable that the progress of the condition will be more rapid. The experimental findings of these two observers bears this out.

The natural corollary of the view held by Coryllos and Birnbaum is that removal of the bronchial occlusion will be of material help in treatment. They have aspirated the bronchial exudate by the bronchoscope from the respiratory passages of infected dogs. They point out that the findings are difficult to interpret on account of the fact that pneumonia in the dog is rapidly fatal and because the resistance of the dog to pneumonic infection is very variable. When aspiration was performed, there was obtained a viscid brown or rusty sputum, comparable to the human pneumonic exudate, but less rich in fibrin. Aspiration failed to produce any pronounced changes in the lobes except possibly in one instance. Nine patients suffering from pneumonia were treated by aspiration. No definite conclusions were formed, but it is stated that no shock nor untoward effect was produced, although some of the patients were old and suffering from toxic absorption. As a rule they claimed to be more or less relieved of the local symptoms by the procedure. The amount aspirated varied from one to twenty cubic centimetres and the exudate was always extremely tenacious and aspirated with difficulty. Coryllos and Birnbaum state that theoretically aspiration would do good only in the early stages of the disease. They compare the exudate in the bronchi to the pus in an abscess and state that the removal of the pus allows healing to occur. The bronchial passages, however, are merely the entrance to a system of very fine ramifications. The alveoli are the structures which most of all require draining. It is difficult to see how the removal of one or even twenty cubic centimetres of viscid secretion from a large bronchus can be expected to do a great deal towards cure, especially as the secretions in the acuter forms of pneumonia are more fibrinous and viscid and hence more difficult to expel.

The crisis in the opinion of Coryllos and Birnbaum is the result of the sudden liberation of a large bronchus and of the consequent drainage of the affected respiratory passages. To accept this view is to ignore the question of the immunological response of the tissues. It may be well to call attention in this connexion to the work of Thomas and Parker which was discussed in these pages as long ago as October, 1920. They showed that when lung punctures were made during the course of the disease, no bacteria were grown some times two days before and some times two days after the crisis. They postulated two mechanisms in the limitation of the infection, a bactericidal and a detoxifying mechanism. They thought that the death of the pneumococcus is not the factor which causes or initiates the crisis and that the antibacterial and detoxifying mechanisms act in a measure independently. Whether this is the true explanation or not need not be argued at the present juncture; it is mentioned in order to show that the crisis can be explained by immunological processes.

Of the ingenuity of the theory advanced by Coryllos and Birnbaum there can be no doubt. It must be owned that the atelectatic conception offers a satisfactory explanation of the lobar nature of the disease and also of the radiographic appearances. Moreover, it is not incompatible with the more orthodox views. Whether the obstruction to the bronchus be primary or secondary, the pneumococci produce a cellular reaction and this supplements the fibrinous exudate in the air passages in arresting the circulation through the lobe. There is much evidence in support of the contention that under the influence of venous stasis a proteolytic action is set up which eventually leads to the breaking down of the cellular and mucoid secretion blocking the air passages. By the time the occlusion to the bronchus is loosened the bacteriolytic action of the tissue fluids has led to the destruction of considerable numbers of pneumococci, so that in the process of resolution the freeing of the bronchus, the readmission of air into the air cells, the absorption of the leucocytic exudate and the dissolution of the pneumococci proceed in rapid succession.

# Abstracts from Current Medical Literature.

## THERAPEUTICS.

### Dextrose.

J. GLASER (*Journal of the American Medical Association*, September 8, 1928) describes the uses and effects of the intramuscular injection of dextrose. The indications for dextrose are numerous — comatose states, toxæmias, pneumonia and enteric fever (in which diseases dextrose by mouth tends to cause tympanites), diarrhœa and generally any condition in which food cannot be taken by mouth. Nassau favoured the use of dextrose also in heart failure, progressive muscular atrophy and *myotonia congenita*. Stamm reported six deaths among twelve infants, the subjects of bronchopneumonia and heart failure, treated with intramuscular injections of dextrose; one contracted gas gangrene at the site of injection. Twenty cubic centimetres of 15% dextrose solution were injected daily for twenty days in one instance with no ill effects. Nassau reported a scleremátous state of the skin after repeated injections; this cleared up when they were discontinued. Several patients with myocardial failure in diphtheria recovered after intramuscular injections of dextrose. In persistent vomiting and acidosis of children good results were obtained. The technique of administration is simple. A 10% solution of dextrose in distilled water or physiological saline solution is injected into the muscles of the anterior and lateral aspect of the thigh; twenty to forty cubic centimetres can be given in infants and up to one hundred cubic centimetres in older children and adults. Absorption is usually complete at the end of an hour. When a 20% solution was injected, too great œdema and tenderness occurred; 10% solutions did not cause great discomfort. In premature and debilitated infants the addition of one part of uncitrated whole blood of the parent to five of dextrose solution appeared to do good more than once. Following intramuscular injections of dextrose the blood sugar rose rapidly in half an hour to 200 to 300 milligrammes per hundred cubic centimetres of blood. This suggests its use in hypoglycæmic conditions. The contraindications to the use of this method are local conditions, hyperglycæmia and bacteriæmia.

### Disinfection of the Biliary Tract.

K. BECKMANN (*Münchener Medizinische Wochenschrift*, November 30, 1928) refers to the disinfection of the gall bladder, especially in typhoid and paratyphoid carriers. After many animal experiments he began to employ a solution of 5% sodium carbonate. This was given in fifty cubic centimetre doses thrice daily by means of a duodenal tube. In addition he used 0·5 gramme in capsule form for oral administration. Five capsules were given thrice daily for three to four days. The results were equally good and were controlled by bacteriological tests of the bile after injection of pituitary extract and the passage of a duodenal sound. Three chronic carriers were successfully treated as well as four persons with inflammatory lesions of the biliary tract due to streptococci, *Bacillus coli* and *Bacillus proteus*. In the latter group the symptoms completely disappeared. He considers that this treatment should be tried for all persons with cholangitis and cholecystitis before an operation is performed.

### The Treatment of Varicose Veins.

G. RIEHL (*Wiener Medizinische Wochenschrift*, October 13, 1928), after discussing the various theories regarding coagulation of the blood and the formation of varicose veins, gives in detail the methods of treatment which he has found to be useful. Patients in the early stages are advised to avoid compression of the veins and also long standing. They are taught how to massage the leg and place a bandage upon it before rising. Rubber stockings and bandages are useless because of maceration of the skin. Unna's formula (gelatine 30, zinc oxide 30, glycerine 50 and water 90) is most useful when the bandage is left on for some time. In the treatment of varicose ulcer Unna's paste is of great value. Chronic granulations and induration of the edges may be treated with carbonic snow or scarification followed by grafting. In the general treatment of varicose veins injection of the lumen plays an important part. After a reference to the earlier use of 1% solution of corrosive sublimate he refers to 15% to 20% solutions of sodium chloride which, although free from toxic effects in general, cause even more necrotic destruction of the connective tissues than corrosive sublimate. Hypertonic solutions of sugar do not cause either general or local reactions and are to be preferred. Dextrose in 66% concentration may be employed, but 50% to 60% solutions of invert sugar act in a more efficient manner. Occasionally 50% solutions of strontium bromide have been used with good results. A ligature is applied above the site of injection and the skin is cleaned with iodine, benzine and alcohol. The vein is massaged to express all blood and the return flow controlled with a finger. The needle is inserted into the collapsed lumen and the fluid injected. The solution is allowed to remain in contact with the vessel walls for three to five minutes before the ligature is removed. The first injection is usually made in the neighbourhood of the knee. Up to fifteen injections may be required before cure results in severe cases. When the condition is refractory to sugar injections, treatment with 15% to 20% solutions of saline to which is added some opium as a sedative is required. Local reaction is usually limited to slight painfulness with occasional redness of the area. Usually the patient can resume his work at once and rest in bed with the leg elevated is forbidden. Injections are repeated at weekly intervals. Contraindications to injection treatment are diabetes, severe cardiac and other general conditions. Pregnancy is not a bar to this treatment.

### Endamœbiasis.

P. W. BROWN (*Annals of Internal Medicine*, August, 1928) discusses the treatment of endamœbiasis. Emetine relieves the acute symptoms, but recurrence is frequent. Auremetine (hydrogen periodide of emetine) given in four 0·06 gramme (one grain) doses by mouth on alternate days for a week give rise to nausea and vomiting and the dose had to be reduced. Stool examinations should be made on three successive days. Normal stools when examined by skilled pathologists are the criterion of cure; there is no other indication of cure. Of 178 patients, 130 were given "Stovarsol" only, 0·75 gramme daily for four or five days; 80% seemed to be cured, but all could not be followed up satisfactorily. Toxic erythema and exfoliative dermatitis occurred in some instances and two deaths were reported. Thirty-nine patients were given "Treparsol" (formyl amide of meta-animo-para-oxyphenyl-arsenic acid), 0·75 gramme every day by mouth for four days. All the arsenic was eliminated by the seventh day. Three courses (twelve tablets in all) were given, four or five days being allowed to elapse between each course. "Treparsol" was thought to be more quickly eliminated than "Stovarsol," hence cumulative effects were less likely. Of the patients 89% appeared cured. Reactions occurred similar to those with "Stovarsol." "Yatren" (Iodo-oxybenzenpyridin sulphonate), three grammes, is given every day by mouth for a week, followed after a week's interval by two or more such doses. An increase of diarrhœa had been noted, but no other untoward symptoms. Of eighteen patients treated eleven were cured and seven relapsed. Bismuth emetine iodide was used as an alternative treatment; six patients were only temporarily relieved by this drug. A combination of emetine and an arsenical preparation gave the best results. Variation and persistence in treatment are advocated to obtain the best results. Amœbic proctitis occurred in forty-one patients in this series; recurrences were more frequent in these patients. Proctoscopic examination revealed the typical amœbic ulcers.

### Ephedrine.

W. A. BLOEDORN AND P. F. DICKENS (*Archives of Internal Medicine*, September, 1928) report a case indicating the danger of the administration of ephedrine in heart failure. Ephedrine stimulates the sympathetic

nervous system, increasing the heart rate and sometimes causing arrhythmia; at first blood pressure is raised, with larger doses the heart's action is depressed and blood pressure falls. It appears to constrict the splanchnic blood vessels, to relax the intestinal muscles, to stimulate the uterus, dilate the bronchi and the pupils. The case reported was that of an adult male who took forty capsules of 0·0243 gramme (three-eighths of a grain) of ephedrine in twenty days for supposed asthma, namely, a smothering sensation at night. When seen, the pulse rate was 160, the heart was dilated and there were obvious signs of decompensation. *Pulsus alternans* was noted. The patient complained of tremor and sweating following the ingestion of ephedrine. Under digitalis normal rhythm and rate were restored. No signs of true bronchial asthma were observed. The condition was one of heart failure from the beginning.

## NEUROLOGY.

### Acute Psychoses Following Influenza.

T. C. GRAVES (*Journal of Neurology and Psychopathology*, October, 1928) quotes Savage who stated that of all the infectious diseases influenza was the most likely to be followed by mental disorder. The most dangerous times are in early youth and at critical periods such as the climacteric and the puerperium. The character of the attack bears no relation to that of the ensuing psychosis. Cases may be grouped into two classes, immediate and delayed. In the immediate group profound general toxæmia may be dominant or toxæmia and mental symptoms may be equal or mental symptoms may be dominant. In the delayed group, after a period of indefinite illness or after an incompletely resolved sinusitis, insomnia and definite mental symptoms arise. In most of these conditions importance is attached to some preexisting septic diseased state in the head, which is aggravated by the influenza and serves to precipitate serious mental disturbance. The prognosis appears to be favourable.

### The Pathogenesis of Disseminated Sclerosis.

G. JERVIS (*Il Cervello*, July, 1928) has inoculated many rabbits with material taken from patients suffering from disseminated sclerosis. Into some animals he injected sclerotic cerebro-spinal fluid endocerebrally, at the same time inoculating them intraperitoneally, intravenously and subcutaneously with sclerotic blood. A number of other animals was submitted to a second complicated inoculation of this nature with corresponding material. Control animals were carefully observed. Although in course of time five animals died after inoculation in none was there proof that death resulted from communicated disease. Full histological examinations both of human material and of tissues from the animals experimented upon failed to disclose any sign of a spirochæte. The writer's conclusion therefore is that the thesis put forward by others to the effect that disseminated sclerosis is a spirochætal disease remains unproven. The paper includes summaries of previous researches on the subject.

### Mental Disorders in Pernicious Anæmia.

P. ÉMILE-WEIL and ROBERT CAHEN (*La Presse Médicale*, July, 1928) indicate that while the organic changes in the nervous system in cases of pernicious and grave anæmia have been fully described, mental disturbances in association therewith have been passed over and yet they are frequent and important. Clinically these disturbances may assume various forms, ranging from simple change of character to actual dementia. In early stages, when the brain is beginning to feel the effect of ischæmia, states of mental depression are the rule. Later, states of excitement are encountered. These may take the form of delirium, simple or with ideas of persecution, and terrifying auditory and visual hallucinations. Other less common forms are simple hallucinatory restlessness, maniacal excitement with confusion and disorientation and a condition resembling Korsakoff's psychosis. These mental disturbances, to be taken as epiphenomena of the anæmia, are attributable to accompanying and well-known disintegrative lesions of the nervous system. With the introduction of new methods for the treatment of anæmia the prognosis is good.

### The Diagnosis of General Paralysis.

JOHN BRANDER (*Journal of Mental Science*, October, 1928) writes a paper because he suspects the diagnosis of general paralysis as today made in many quarters and in consequence doubts the validity of many published statistics, especially those bearing on curability. From the time of Esquirol who, by the way, stated that he saw only two recoveries, this disease has been readily and accurately diagnosed not only by medical practitioners, but by mental hospital attendants; indeed it has been remarked: "You do not diagnose general paralysis; you sense it." The diagnostic criteria of the disease and likewise its fatal prognosis were established years before the discoveries of Argyll Robertson and Wassermann. But times have changed and today it seems that conditions are brought within the category which the older psychiatrists would certainly not accept as general paralysis. When the relation between syphilis and general paralysis became proven, the introduction of the Wassermann test appeared likely to clinch and clarify the diagnosis. Instead it brought further faultiness in the shape of the era of so-called mixed forms of cerebro-spinal syphilis. It is these mixed forms which have provided the existing confusion and it is between these and typical general paralysis that it is fundamentally necessary to maintain a definite distinction.

### Tumours of the Fourth Ventricle.

LUDO VAN BOGAERT and PAUL MARTIN (*Revue Neurologique*, September, 1928) record twelve tumours of the fourth ventricle. In the matter of diagnosis they emphasize what is called the "cerebellar syndrome of the middle line." This syndrome is characterized by sudden onset, due apparently to hydraulic disturbance in the posterior cerebral fossa; violent paroxysmal headache; tonic spasm of neck muscles resulting in an antalgic attitude of hyperflexion; aggravation by change of position; vertigo and nystagmus; cardio-respiratory irregularity; equilibriatory disturbance; muscular hypotonia; and a papillœdema which is precocious. The phase of decline is characterized by further bulbar trouble and death may be sudden. By this syndrome along with evidence of blocking of the aqueduct of Sylvius, proved by Queckenstedt's test, ventricular tumours are to be distinguished from tumours of the cerebellar hemispheres and other subtentorial growths. Although the prognosis is most grave, good results do sometimes follow surgical treatment, early diagnosis being an important requirement.

### Juvenile Behaviour Disorders After Encephalitis.

T. R. HILL (*Journal of Neurology and Psychopathology*, July, 1928) bases a paper on observations made in the Post-Encephalitis Lethargica Unit of the Metropolitan Asylums Board, London. In describing the clinical features of post-encephalitic conditions he states that the children have a preserved intelligence and clear insight into their condition, realizing the error of their conduct. The latter is compulsive and consists of actions of a crude, primitive and instinctive type. Its compulsiveness is regarded as being due to an overpowering increase in the pleasure that normally comes of obeying instinctive tendencies and in the displeasure that normally comes of obstructing them. The pathology may be an organic encephalitic process causing exaltation of thalamic function, either by thalamic release through loss of cortical inhibition or by chronic irritation and excitation. This, the writer thinks, is supported by (i) the known incidence of the encephalitic virus on the thalamo-striatal level and (ii) the preservation of memory and intelligence, indicating that the cortex cannot be seriously involved. Also the thalamus probably has some action in the origination of primitive and instinctive behaviour.

# British Medical Association News.

## SCIENTIFIC.

A MEETING OF THE SECTION OF NEUROLOGY AND PSYCHIATRY OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Red Cross Hall, Callan Park, on November 8, 1928, DR. H. C. McDOUALL in the chair. The meeting took the form of a series of clinical demonstrations.

PROFESSOR W. S. DAWSON, at the beginning of the demonstration, stated that the patients shown at the meeting were intended as representative of the different types of neurological and mental patients dealt with in the Repatriation Block. Many of the conditions were not in themselves distinctive, but, taken as a series, made a good demonstration of post-war neuroses and psychoses.

### Traumatic Paraplegia.

PROFESSOR DAWSON showed a male patient aged thirty-three years who had received a wound on the left side of the chest from a shrapnel bullet on August 11, 1916. He had immediately lost all power in his legs, with complete anæsthesia from the umbilicus downwards, with incontinence of urine and fæces.

X ray examination showed the bullet to be embedded in the neural canal at the level of the first and second lumbar vertebræ. He had regained a certain amount of the use of his legs and became able to walk with the aid of sticks. He had remained in military hospitals and at home until June 29, 1928, when he became mentally confused and depraved in habits.

The lower limbs were flaccid and both feet were inverted. There was very slight power of movement in the toes. His mental condition did not permit of an accurate estimation of the sensory functions.

DR. A. W. CAMPBELL said that he considered that the condition was a typical example of a *cauda equina* lesion and that he would expect to find a typical saddle shaped area of anæsthesia.

### Traumatic Epilepsy.

Professor Dawson's second patient was a male, aged thirty-five years, who had been wounded in the parietal region in September, 1917. He had at once commenced to have severe fits. In November, 1918, he had been operated on in the hope that the fits might be relieved and in six months he had improved very considerably and had been able to return to work. After twelve months the fits had recurred and he had gone to Broughton Hall. At another operation four foreign bodies had been removed. He had again improved and had worked until 1924, when he had again broken down and been admitted to Callan Park. On October 12, 1927, he had had a typical seizure (major), but had not had one since.

His character was that of a typical epileptic. He made false accusations against the attendants, was unstable and egotistical with the delusion that he could cure every disease. He had ideas of persecution, was irritable, bad tempered with occasional outbursts of dangerous violence.

Professor Dawson's next patient was a male, aged forty-two years, who had received a head wound in the war. He had been admitted on May 4, 1926. He gave a history of epileptic fits, especially associated with bouts of alcoholic over-indulgence. Fits in the hospital were infrequent, about once a month, followed by much restlessness and confusion. He was childish, dull and incapable of mental effort and could neither read nor write. His memory was poor. His speech was almost totally incoherent. He had a large depressed area in the left temporal region of the skull.

### Alcoholic Neuritis.

Professor Dawson also showed a male, aged twenty-nine years, who had been admitted to Broughton Hall in November, 1916, and whose condition had been diagnosed as *melancholia à potu*. He had been drinking paraldehyde in large doses.

He had been admitted to Callan Park as a voluntary patient in January, 1928, after drinking heavily of paraldehyde and alcohol. He complained of neuritis of the left forearm and hand of six months' duration. Four years previously he had had a similar seizure from which he recovered. Latterly he had noticed a wasting of the small muscles of his left hand. He complained of tenderness and pain in the left arm and hand; particularly in the ulnar distribution. The grip of the left hand was weak. The interosseous muscles were wasted. There were fine tremors in both hands, particularly the left. In childhood he had sustained a fracture of the left elbow, where there was a deformity of the ulna bone. He was not clear whether the wasting of the left hand dated from that injury or was more recent, but it appeared that alcohol was responsible for the pain and for impairment of muscular power.

DR. A. W. CAMPBELL said that he thought that an ulnar lesion fairly early in life might have caused the condition.

### Huntington's Chorea.

DR. J. A. L. WALLACE showed a single man, aged forty-four years, who had been admitted on May 5, 1926, suffering from Huntington's chorea. The patient was a native of South Africa and a customs official; there was no hereditary history of mental disease. He had suffered from enteric fever at the Boer war. He had been wounded in the right arm in the European war. He had paralysis of the radial nerve and wrist drop, though the nerve had been sutured. His ship had been torpedoed and he had suffered from mental shock when other passengers, including women, were drowned. He had gone back to South Africa after the war, had got sick leave and come to New South Wales. Choreic symptoms had been present since the war. He had got ideas that he was treated badly by his family and had also been drinking.

This patient had been shown at a clinical meeting at Sydney Hospital and also later at a meeting of the New South Wales Branch while he was in the earlier stages of chorea.

Huntington's chorea could often be traced through generations. It consisted of choreic movement with ataxy, associated with mental degeneration. Histologically there was degeneration of the cells of the cerebral cortex and basal ganglia with neuroglial proliferation and meningeal thickening.

### Paranoia with Chronic Hypomania.

Dr. Wallace also showed a man aged sixty-two years, married, a native of Tasmania, an accountant, who had been admitted on January 9, 1928. His father was a "bit of a rover." The patient was the eleventh of a family of twelve. He had been in France, but was neither gassed nor wounded. He had been ill and discharged "unfit," with a weekly pension of fifteen shillings. Since the war he had suffered from heart weakness and myalgia of the legs. He had not had regular work since the war. He had worked at "Base Records" and then at a soap factory. He had written a book on economics but this had failed to be accepted by publishers. About two months before admission he had begun to write abusive letters to certain high political officials. The following was an example of the patient's effusions:

"BLOOD MONEY."

*Addressed to "Directors of Callan Park."*

"Blood money is the meanest pay
The serf may earn, ignoble fee
And this blood money won through me
That feeds lost souls from far away."

"I laugh in scorn at those who run
These modern catacombs of Rome
In this the country I call home
And fought for till the war was done."

"Mean souled and petty worms are they
To hide their sordid perfidy
Have buried here a man like me
Whose love of right no power can sway."

"Humble deciple of Courageous Tennyson.
Ward 6, Aug. 4th, 1928.
Rolled into a cigarette this ought to taste good."

The patient was suffering from paranoia with chronic hypomania. He had been permitted to go on leave on March 5, 1928, and had returned a month later on account of causing annoyance by writing letters to various politicians. He had again been permitted to go on leave on August 22, 1928, and had come back a fortnight later arrested by the police for having written offensive letters to the Premier. The patient resented being detained and one reason why he had at first objected was that he had contracted to give a lecture at the Trades Hall and had had to cancel this.

### Aphonia.

Dr. Wallace's third patient was a man, aged forty-two years, a motor engineer, a voluntary patient who had been admitted on May 6, 1926. He had been in Number 13 Australian Auxiliary Hospital in May, 1918, and his condition had been regarded as hysterical aphonia. He had been on active service in France, but not in action. He had driven a Red Cross lorry, had got ill with pain in chest. His illness had been diagnosed later as chronic fibroid phthisis. The patient had not spoken, he had written everything down, sometimes he would not write. He had worked well and had been able to speak, as he had been heard speaking. He had been discharged from Number 13 Australian Auxiliary Hospital on January 27, 1919. He had gone to Bondi for a few weeks and then to Lane Cove House for three years. The Repatriation Department had then placed him in "Russellea" for four months and for a period of three months he looked after a poultry farm at Mitford. He had been there twenty months, had then suffered from dysentery and been in Auburn Hospital. Then he had been in Lidcombe for a short time and then had gone to Wagga and got a light job at a theatre, collecting tickets and handing out pass-out checks. He had been there four months, but it had got too cold for him and he had returned to Sydney. He had boarded at Woollahra for about two years. He had finally gone to Randwick and had asked if he could come back to the Repatriation Section of Callan Park for treatment. The patient had frequently been granted leave, but later this had had to be curtailed owing to his obtaining drink. He never spoke, but invariably wrote down all he had to say. He was able to work usefully, but if allowed away from institutional care could not be prevented from drinking to excess and very little affected him.

### Neurasthenia.

DR. G. B. R. WOOSTER showed a patient who was suffering from neurasthenia with some depression. The patient was fifty-three years old, single and an accountant by profession. He had been admitted on June 18, 1928. He had had nerve trouble for ten years or more. He complained of a feeling of continuous lassitude and lack of energy. He had constant occipital headache and eyesight impairment. He had a severe pain in the abdomen for which no adequate reason could be found. He was sleepless, moody, irritable and full of complaints. He could not concentrate, was distractible and if he read a book, all the words became jumbled up and senseless. He was much improved since coming to hospital.

### Idiopathic Epilepsy.

Dr. Wooster's second patient was a man, aged thirty-one years, single, a fitter. He suffered from idiopathic epilepsy. He had been in hospital for the previous two years and before that in Broughton Hall for a short period. He had had fits since the age of seven. Despite this he had managed to reach France during the war, but had developed influenza. He had been sent to hospital, where his epilepsy was evidently discovered and he had been invalided home. He had a typical and severe epileptic fit fairly frequently, more so of recent years and if he was not having medical treatment. He had an aura sometimes, "a funny feeling in the head." He passed urine and fell on his head, often inflicting injuries. For this reason he wore a padded hat. He had deteriorated, was dull, childish, bradyphrenic with impaired memory and typical slow deliberate speech of the epileptic.

### Manic Depressive Insanity.

Dr. Wooster also showed a patient, aged forty-eight years, who was suffering from manic depressive insanity. The patient was a married man and had two children. He was a clerk and had first been admitted on November 2, 1920. There had been a history of potus and of threatening violence to his wife. He had been maniacal, restless and had required a single room. He had again been admitted on December 18, 1925. He had been restless, elated, self-satisfied on admission. He had become well on May 10, 1926, and then manic again. He had become well on November 6, 1927, and hypochondriacal and lacking in interest on December 30, 1927. He had then become maniacal again, destructive, dirty, restless, noisy, troublesome. Sudden improvement had occurred in April, 1928, and at the time of the meeting he was quite well again. Though he was never severely depressed, it was obvious to nurses who knew him well, that he had definite periods of depression of a mild character. These usually occurred just before a maniacal phase made itself apparent.

### Dementia Præcox.

DR. J. M. RAINBOW showed a patient who was suffering from *dementia præcox*. There was a family history of insanity, the uncle and sister being affected. The patient's first admission had been on April 23, 1920. At this time he had been twenty-four years of age. His blood had not reacted to the Wassermann test and he had been returned from war service two years. He had been evacuated with shell shock and symptoms of delusional melancholia, thought to be due to *dementia præcox*. He had been inclined to be reticent.

On October 10, 1927, he had manifested catatonic symptoms; had adopted grotesque attitudes and puffed out his chest. On September 18, 1925, he had been childish and irresponsible, restless and delusional. He had manifested schizophrenic and catatonic reactions. He had exhibited *cerea flexibilitas*. On March 23, 1927, he had been mute, manneristic and idle and on June 30, 1928, mute, manneristic and demented, but in excellent health.

### Melancholia à Potu.

Dr. Wooster also showed a man, aged forty-two years, who had first been admitted on March 17, 1924. He had yielded no reaction to the Wassermann test. He had not worked for years owing to auricular fibrillation. He gave a history of alcoholic excess, was dull, unsociable and suspicious and had delusions of persecution. His condition had been diagnosed as *melancholia à potu*. He had seen war service abroad as a private.

The following details from his record were quoted to indicate the patient's instability:

April 1, 1925: On leave and returned alcoholic.
May 21, 1925: On leave and returned alcoholic.
August 25, 1925: On leave and returned alcoholic.
February 5, 1926: Readmitted in a state of alcoholic confusion.
December 19, 1926: Recovered.
April 26, 1926: Returned from leave, intoxicated.
October 22, 1926: Returned from leave, intoxicated.
July 14, 1927: Readmitted, depressed and alcoholic.
July 26, 1927: Not depressed, well.
October 15, 1927: Readmitted alcoholic.
February 3, 1928: Absconded.
February 4, 1928: Readmitted, alcoholic.
April 25, 1928: Apparently normal mentally, but lacks control. Defective moral sense (pawns mother's clothes for drink).
August 30, 1928: Good worker on dray, but goes to hotel (absent without leave). Got drunk, removed to closed ward.

### General Paralysis of the Insane.

DR. C. HENRY showed four patients suffering from general paralysis of the insane, illustrating the improvement after malarial treatment.

The first patient had been admitted to Callan Park on July 14, 1913, when the differential diagnosis had been given as *dementia præcox* or general paralysis of the insane. He had been dull, "silly," suicidal and of poor intelligence. The blood had reacted to the Wassermann test. On December 27, 1913, he had been discharged and later had enlisted in the Australian Imperial Forces and had been gassed and affected by shell shock in France. On September 15, 1921, he had been admitted again to Callan Park. He had been noisy, restless, idle, incoherent, troublesome, delusional, careless and untidy, in which state he had remained for nearly seven years.

On March 26, 1928, he had received an intravenous injection of malarial blood and had been allowed the usual course of rigors. Immediately after this he had begun to improve and at the time of the meeting was a cheerful, reliable worker in the hospital.

The second patient had served in France from 1915 to 1919 and had been gassed. On July 11, 1927, he had been admitted to Orange Mental Hospital unconscious, in a fit due to general paralysis. He had been transferred to Callan Park on July 18, 1927, and had been disorientated, dull and demented, with faulty habits and gangrene of one toe.

The Lange test had yielded the following curve: 4433210000; lymphocytes had numbered 65 cells per cubic millimetre, and a strongly positive response had been obtained to the Wassermann test.

He had been infected with malaria on June 3, 1927, and had had eleven rigors, his temperature reaching 40·6° C. (105° F). In two months he had improved very considerably, being agreeable and of moderate intelligence, in robust bodily health and his serological condition had improved also. The Lange test had yielded the following curve: 4212000000, with three cells per cubic millimetre and indefinite result to the Wassermann test.

The third patient had been admitted on August 23, 1927, in a very delusional state, euphoric and quite unable to realize his position. The Lange test had yielded the following curve: 5433221000.

He had rapidly improved both mentally and physically after treatment with malaria, regaining insight into his position and the Lange test had yielded the following curve: $31\frac{1}{2}\frac{1}{2}\frac{1}{2}0000$.

The fourth patient had been admitted to Callan Park on September 15, 1922, in an idle, delusional, aggressive and noisy state. He had remained a very refractory patient for six years until treated with malaria, when he had at once improved and was now a quiet orderly worker.

Dr. Henry said that of the sixty-eight patients he had treated by malaria therapy in the previous eighteen months, thirty-three had shown improvement, among them several in whom the improvement was much more pronounced even than in the patients shown.

DR. H. C. MCDOUALL said that whatever one might think of the treatment of general paralytics with malaria in only prolonging their lives, even although no economic end might be gained, it was the first duty of a medical practitioner to prolong life.

He referred to the different treatment of general paralysis at an earlier date than when he was Medical Superintendent of the Gladesville Hospital and since then by "Sarvarsan" and to the practically complete failure of these forms of treatment.

DR. KENNETH SMITH said that, although not a member of the Section, he was very pleased to be present, as for some two years he had had experience of these patients from an administrative point of view. He was constantly being asked by the Executive Staff of the Repatriation Department what treatment was being given to the patients in the Repatriation Block at Callan Park. As members of the Section could see, the Repatriation Commission had been able to erect buildings for the accommodation of their patients which differed somewhat from the usual type of mental hospital buildings and approximated more to hospital wards. He was pleased to find that the methods of treatment in use were as advanced as in other parts of the world and although the prognosis as regards recovery was very bad in many of the patients, yet by suitable therapeutic methods many patients might recover sufficiently to enable them to live outside an institution. He pointed out the great difference that the pension made in those conditions accepted as attributable to war service and stated that such mentally afflicted patients were able by means of the pension to provide for any number of children, as every child of a pensioner, whenever born, was pensionable. It was possible that this provision might result in the birth of many children with bad heredity who thus became potential future patients of mental hospitals.

DR. J. A. L. WALLACE referred to Dr. Henry's patients with general paralysis and said that he was convinced that malarial treatment had undoubtedly prolonged their lives. He instanced one of the earlier patients who had been treated with a falciparum strain of malaria, who after a series of severe rigors had eventually recovered to such a degree that he was earning a competent livelihood. Apart from this particular case, undoubtedly the malarial treatment had modified the usual course of the disease and prolonged the lives of the patients. He stated that in the small world of the mental hospital they were satisfied that if the patient could not be cured, he could be made to occupy himself usefully. He then referred to occupational therapy in the form of raffia work in which a number of Dr. Kenneth Smith's repatriation patients was engaged with highly beneficial results. Apart from these patients quite a number of general paralytics was useful hospital workers.

# Medical Societies.

## CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN, BRISBANE.

A MEETING OF THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN, BRISBANE, was held on August 30, 1928, DR. J. LOCKHART GIBSON in the chair.

### Œdema Neonatorum.

DR. J. G. AVERY presented a child who had been born at the Lady Bowen's Hospital nineteen days previously. It had been brought to the out-patient department twelve days before on account of a rash over the body. The rash had consisted of papules and vesicles and had been bilateral. There had been no pitting on pressure and no itching. The child had cried when it was being bathed. There had been hard, red and tender indurated areas on the thighs.

The mother had had seven children, two of whom had been still-born. She had had no miscarriages. All the other children were healthy. The mother's serum, that of the patient and that of her two sisters had been subjected to the Wassermann test. No reaction had been obtained.

A blood examination had been carried out. The red blood corpuscles numbered 6,090,000 per cubic millimetre; the leucocytes 6,400, the hæmoglobin value was 120% and the colour index 1·0. There were no abnormal red cells. Of the leucocytes 54% were neutrophile cells, 40% lymphocytes and 6% eosinophile cells. On August 20, 1928, there were 5,600,000 red blood corpuscles, 8,000 leucocytes of which 39% were neutrophile cells, 53% lymphocytes, 6% large mononuclear cells and 2% eosinophile cells. The hæmoglobin value was 100% and the colour index 0·9. There were no abnormal red cells. Dr. Avery suggested that the diagnosis lay between *œdema neonatorum* and *sclerema neonatorum*.

### Tumour of the Groin.

Dr. Avery's second patient was a boy, aged ten years, who had been admitted to the out-patient department on August 20, 1928, on account of a lump in the groin which had been present for three years. The swelling had been soft and fluctuant and appeared to be in two parts, one

above and one below Poupart's ligament. The swelling had been over the femoral vessels and there had been an impulse on coughing over the upper part of the swelling, but not over the lower part. Both parts had been easily reducible. The lump had not caused the child any trouble except when it disappeared. The child had cried with pain until the swelling reappeared. He had been admitted to a hospital in order that an examination of his blood could be carried out. Filaria had been found in the blood.

### Compound Fracture of the Humerus.

DR. A. V. MEEHAN exhibited a child, aged eight years, who had been admitted on June 11, 1928, from the country with a compound fracture of the right humerus. The wound had been cleaned and a fragment of bone projecting through the skin had been gently pushed back. On admission the arm had been swollen and the discharge had been offensive. Spirit dressings had been applied. On June 20 the dressings had been removed and the wound cleaned with spirit. The fracture had been reduced as far as possible under an anæsthetic. A Jones's metal splint had then been applied with traction. A skiagram had been taken and had revealed a comminuted fracture above the condyles of the humerus with the condyles in fair position. The patient had been distinctly ill and the temperature had been raised. The dressing had therefore been loosened at intervals. On July 20 a skiagram had been taken and the radiographer had reported that the position had not been good. On August 11 the upper end of the lower fragment had been seen in a skiagram to be in good position, while the lower end of the upper fragment was still directed forwards. Union had taken place between the lower fragment and the posterior surface of the upper fragment. The humerus had healed in good position with firm bone, but a spur of bone had arisen from the anterior surface of the upper fragment and had prevented free flexion. Dr. Meehan had removed the spur at an operation on August 29, 1928. The arm had been put up in metal splints with the elbow at right angles.

### Spindle-celled Sarcoma.

DR. G. P. DIXON showed a child, aged ten years, who had been admitted to hospital on July 24, 1928, with a lesion of the left foot. The child had been kicked while playing football three weeks previously. He had been healthy before and had not had any other injury. The foot had swelled. It had been incised eight weeks before admission and again seven days before. When seen there was a raised, red area on the lateral margin of the foot with a pustular surface. There had also been a lump in the left inguinal region. Dr. Dixon had amputated the foot above the ankle joint. Part of the tumour had been examined microscopically. It was found to be a small spindle-celled sarcoma with mucoid and myxomatous degeneration. Abundant secondary deposits had been discovered in the gland removed from the groin. During convalescence the patient had developed a paralysis of the left external rectus muscle which had not cleared up.

### Bacterial Endocarditis.

DR. GAVIN H. CAMERON presented a boy, aged ten years, who had been admitted to hospital on July 25, 1928, on account of an illness diagnosed as pneumonia. The child had not been well for ten days. Two days after the onset he had complained of pain in the abdomen, but he had not vomited. The pain had wandered from the abdomen to the knees, elbows and arms and when admitted was in the left elbow. His breathing had been laboured and shallow and the child had looked very ill and grey. There had been a blotchy rash over the body. The tongue had been dirty, but the throat had appeared to be normal. The percussion note over the lungs was normal. The breath sounds at the base of the left lung had been diminished, but no adventitious sound had been present. The heart sounds at the apex had been normal, but the first sound at the pulmonary orifice had been absent. After admission the child had had remittent fever. On July 26 a skiagram revealed that the bases of both lungs were opaque. On July 30 a definite systolic murmur was detected at the apex. Crepitations had persisted. On August 5 a skiagram revealed a very large cardiac shadow. No reaction had been obtained to the Wassermann test. On August 7 a blood count had revealed that there were 4,410,000 erythrocytes per cubic millimetre and 10,000 leucocytes, of which 65% were neutrophile cells, 30% lymphocytes, 4% large mononuclear leucocytes and 1% eosinophile cells. The hæmoglobin value was 88% and the colour index 1·0. No bacteria could be grown from the blood. On August 17 a to and fro murmur had been heard at the fourth interspace. The murmur had varied considerably. Occasionally a murmur had been heard at the base. The diagnosis was bacterial endocarditis and pericarditis.

### Pernicious Anæmia.

DR. S. F. MCDONALD dealt with the case of a male, aged seven years and five months. He had been admitted to hospital on June 11, 1928, with a history of gradually increasing illness for three months. He had had pain in his limbs and joints and increasing pallor. At the time of his admission his spleen had been felt below the costal margin, but this had subsequently disappeared. No signs had been detected in the chest. On June 11 the blood count had been: red blood corpuscles 2,500,000, leucocytes 5,400, of which 34% were neutrophile cells, 62% lymphocytes, 2% large mononuclear cells and 2% eosinophile cells. Punctate basophilia, polychromasia, microcytosis and nucleated red corpuscles were present. A test meal had been given and it was discovered that there was an absence of hydrochloric acid. A skiagraphic examination had revealed atrophy of bones, an absorption line above the epiphyses, lack of calcium in the diaphyses. Dr. McDonald called attention to the pathological nature of these changes. The Price-Jones curve had not borne out the diagnosis of pernicious anæmia. The child had been fed on 110 grammes of liver and had received 3·5 cubic centimetres (one drachm) of hydrochloric acid three times a day. Improvement had followed. On August 27 the blood count resulted in the following figures: red blood corpuscles 3,150,000, leucocytes 7,000 of which 42% were neutrophile cells, 54% lymphocytes, 1% large mononuclear cells and 3% eosinophile cells. The hæmoglobin value was 60% and the colour index 0·9. There was some polychromasia, some anisocytosis and some punctate basophilia.

### Cerebral Tumour.

Dr. S. F. McDonald next called attention to a girl, aged eleven years and five months, who had been ill for twelve months. Her symptoms had included very severe headache, earache, vomiting and later paresis of the right eyelid and left side of the mouth. Still later paresis of the left external rectus muscle had appeared. On admission it had been found that papillœdema was present. The child had been totally deaf. No galvanic reaction was obtained and the caloric test yielded no response. The drums appeared natural. Irregular nystagmus had been noted. Fourteen days before the meeting the patient had shown a tendency to walk to the left. The blood and the cerebro-spinal fluid failed to deflect complement in the Wassermann test. A lymphocytosis was present. Lumbar puncture had been performed on several occasions. The diagnosis lay between cerebral tumour and cerebral syphilis.

Dr. S. F. McDonald also showed a female child, aged five years, who was supposed to have had a cold five weeks previously. This had been followed by headaches and the appearance of squint. Papillœdema had been discovered. The diagnosis of cerebral tumour had been made, but the condition was clearing up in response to repeated lumbar punctures.

### Sarcoma of the Orbit.

DR. J. LOCKHART GIBSON related the case of a child six months of age who had been seen when there was considerable proptosis of the left eye. The child appeared to be in good health and not to suffer any pain. Ophthalmoscopic examination had revealed white discs. The pupils were dilated and insensitive. The whole of the optic nerve had been affected. Dr. Lockhart Gibson had made the diagnosis of sarcoma involving the optic nerve. He had

enucleated the eye and had cleared out the orbit. The growth had extended as far as the optic foramen. A piece of the optic nerve had been removed by traction. Microscopic examination of the tumour had shown it to be a spindle-celled sarcoma. Five milligrammes of radium had been inserted into the back of the orbit and left there for sixty hours. No recurrence had taken place, although six months had elapsed since the operation.

## THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING OF THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA was held at the University of Adelaide on October 5, 1928.

### Blood Calcium in Women.

DR. R. F. MATTERS discussed the results of further experiments on the blood calcium-content of women both during pregnancy and in the various phases of the menstrual cycle. The results were derived from some three hundred estimations of the blood calcium of about seventy women. In the menstrual cycle it had been found on an average that there was a curve the highest point of which was four days before the onset of menstruation. From here a rapid fall of blood calcium took place until the fourth day after the onset of menstruation. The fall still continued after this, but very gradually until the sixth day, when a gradual rise took place, increasing in rapidity until the zenith, four days before the onset of menstruation. Accurate graph curves were shown.

Patients had been examined on exactly the same day of the cycle on two and three occasions, but with some months' interval and these results had all been coincident, showing that the blood serum estimations had been accurate.

The blood calcium during pregnancy was found to decrease as pregnancy advanced which was in accord with the work of Mirvish and Bosman, who showed that œstrin injected into rabbits and human beings produced a fall of blood calcium. Dr. Matters related the fall of blood calcium to the increase of the ovarian hormone as shown by the test of Siddal. The Siddal test for pregnancy which had been mentioned, was dependent upon the circulation of the follicular hormone in the maternal blood stream, the hormone being mainly secreted in the placenta.

The method employed was the withdrawal of the blood to be tested into a sterile test tube. After the serum had separated one cubic centimetre was given to a mouse subcutaneously. This same serum was injected daily into the same mouse for four days. The mouse was destroyed on the fifth day and weighed. The uterus and adnexa were then removed and weighed. The first weight in milligrammes divided by the second gave a result which was in the region of one to two hundred if the serum was from a pregnant woman. Otherwise from males and non-pregnant women the result was in the region of 900 to 1,000.

Twenty-five patients had been tested and gave this result. With the alteration of blood calcium during the menstrual phase it was thought that the follicular hormone intensity varied considerably and that it was possible that influence upon other glands was appreciable. With this in view small hypodermic doses of adrenalin had been given during the menstrual cycle and thirty-four patients had been so tested. The results were that on an average the adrenalin produced vertigo, hot flushes, and headaches between the twelfth and first day before the onset of menstruation, after which the result was nil or the symptoms occurred in a minor degree.

A number of estimations of the basal metabolic rate had been made both during pregnancy and in the menstrual cycle and there appeared generally to have been thyreoid alterations, but sufficient estimations had not been made to warrant any dogmatic statement.

DR. L. B. BULL queried the accuracy of the analyses for calcium made in the Adelaide Hospital Laboratory for Dr. Matters.

Dr. Matters pointed out, however, that the errors in the analyses, although affecting the absolute figure, did not affect the relative figures in the cases questioned.

DR. J. G. SLEEMAN asked why the flushes occurred with the adrenalin and not the normal cold feeling.

Dr. Matters stated that it was a question of invalence between the glands and that the reaction obtained was not therefore the normal vaso-constriction one.

DR. C. S. HICKS, in reply to Dr. Sleeman's drawing attention to the variability of the result obtained, pointed out that the results would be largely affected by the preponderating effect of sympathetic or parasympathetic systems and that these would be individual and show considerable alterations during the period.

MR. H. E. LINES referred to persons with enlarged thyreoids who lived on land rich in mineral salt, and therefore in potassium iodide, but with phosphate deficiency, the phosphate deficiency implying also a calcium deficiency.

DR. C. S. HICKS referred to goitres in sheep on the coasts of the Canterbury Plains which were getting quite ample iodine intake.

### Nutritional Disorder in Ewes.

DR. L. B. BULL gave an account of ewes living on soursobs (oxalis). The animals were very weak and one could progress only on its knees; another had been down for two weeks. Spasms of certain groups of muscles of legs and neck were shown. The tetanus condition was suspected to be due to calcium disorder. Another farmer had a group of nine hundred young ewes similarly affected of which at the date of examination eighty-five had succumbed. They had been put on the feed in July and the tetany had appeared some weeks later; death had soon supervened in most cases without spasms. In these cases at *post mortem* examination the serous cavities were found to be filled with fluid, the kidneys white and small. The kidneys were extensively fibrosed, glomeruli normal, tubules mostly affected, replaced largely by fibrous tissue. Crystals were noted permeating the kidney tissue; they were soluble in hydrochloric acid.

In one instance three grammes of a kidney on analysis had contained fifty milligrammes of oxalic acid; in another case seventy-five milligrammes. It was computed that one gramme of oxalic acid was present per kidney. The serous exudate was considered to be due to calcium deficiency in the cells. Blood calcium was normal. The clotting time was normal.

Blood from an animal in spasm and taking six hours to clot, showed a normal calcium figure. One animal had been treated with parathyreoid extract (Oppenheimer and Eli Lilly used) and no effect had been obtained. The urine had been alkaline to phenolphthalein and this had suggested an alkalosis. Ammonium chloride two grammes and calcium chloride two grammes had been given and in twenty-four hours the urine had been acid and contained 0·12% ammonia and 1·8% urea, being a nitrogen ratio of 1 : 8·5. The animal had improved and the dose had been dropped to two grammes of calcium chloride three times a day.

A demonstration of sections of the kidney was examined. The crystal deposits were obvious.

## CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN.

A MEETING OF THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN was held at the Hospital for Sick Children, Brisbane, on November 22, 1928, Dr. J. LOCKHART GIBSON in the chair.

### Cœliac Disease.

DR. SHIRLEY LANE showed a male child, aged seven years, who was suffering from cœliac disease. He had attended

Dr. Lane's out-patients' clinic two years previously with a history of having a voracious appetite and passing large foul stools. He had been found to be undersized, to have a large protuberant abdomen. The stools had been foul and loose and full of fat floating to the top. The spleen and the liver had been normal in size and there had been flatus present in the abdomen. X ray examination had revealed a block at the ileo-caecal valve. On a fat-free diet the stools had cleared for a while. The child had then contracted chicken pox and impetigo and had become emaciated and pale and the skin had become dry. The teeth and tonsils had been septic.

In September, 1927, there had been no improvement, though the teeth and tonsils had been treated.

The child had been seen one month before the meeting and there had been a striking change; the abdomen had decreased in size and in the year the child had grown 3·75 centimetres (one and a half inches). The mother said that the stools were almost normal. The diet had consisted of skim milk, bread, no butter or whole milk, no fat or yolk of egg. Pancreas had been tried, but the child had refused to take it.

### Syphilitic Dactylitis.

DR. S. F. MACDONALD showed a female patient aged six years and eleven months. The history was that there had been swelling of the phalangeal joints of the right hand and of the left foot. The swelling of the left foot had disappeared, but in the right hand there was a definite swelling, gradually getting worse and varying in intensity, being generally better in the morning and becoming worse during the day. The only abnormality found on a thorough examination was a strong response to the Wassermann test. X ray examination had revealed no abnormality. There had been a slight rise of temperature on two occasions, otherwise the temperature was normal.

The diagnosis rested between syphilitc dactylitis and an early manifestation of Still's disease. The outlook in the first condition was good, in the second hopeless.

Still's disease could begin in this way with very little swelling which subsided and then appeared elsewhere, but in this patient there was no enlargement of the glands and the spleen was palpable.

The diagnosis would probably be settled by the condition cleaning up with vigorous antisyphilitic treatment.

### Chronic Osteomyelitis.

DR. A. V. MEEHAN showed a male patient in order to illustrate his method of treatment in cases of chronic osteomyelitis. This patient had reached the chronic stage of the disease, the focus in the femur being of three years' and that in the humerus of twenty-one months' duration.

There was a huge cavity in the femur and the treatment adopted had been to open up and clean out the cavity. It had been impossible to convert it into a groove, as it had been too extensive. There had been extensive chiselling of the bone and most of the femur had been removed and healthy bone had been left all round. The operation had been very radical and had been done with a tourniquet applied so that there had not been so much shock. The wound had then been packed with vaseline gauze and a plaster applied which had been left on for three weeks. As the operation had been so radical there had been a fair amount of bleeding and the blood had oozed through the plaster. This was the only disadvantage of the treatment—the plaster had become foul-smelling. Even though the oozing had been extensive, he had not had to remove the plaster.

The first dressing had been done three weeks after operation and when the plaster was removed and the gauze taken out, healthy granulations had been found. The cavity had again been packed with vaseline gauze and dressed once a week.

Dr. Meehan did not consider that, even in an acute case, it was necessary to dress the wound every day. The arm had been treated in the same way as the leg fourteen days before and was practically healed. Skiagrams of the cavity were shown.

### Pituitary Lesion.

Dr. A. V. Meehan showed a boy, aged ten years, who had been brought to the out-patient department on account of "weak ankles." He had been found to have long thin feet, had walked in eversion and *valgus*. On examination of the other limbs and of the bony skeleton there had been found a general elongation of all long bones. He had had trouble with his eyes but on being fitted with glasses had been quite smart at school. He was probably an example of a patient with some lesion of the pituitary body and it was decided that no treatment would be of any avail.

### Lesion of Lymphatic System.

Dr. Meehan's third patient was a child which was suffering from hemihypertrophy of one side with accompanying nævi on that side. The parents stated that the hypertrophy had been proportionately the same since birth and that the nævus also had been present at birth and had grown correspondingly. It was presumed that the child was the subject of some disturbance of the lymphatic system.

### Pink Disease.

DR. H. MATHEWSON showed a male patient aged twenty-one months, who was suffering from typical pink disease. He manifested redness and coldness of the nose, œdema and coldness of the limbs, erythema over the body, was irritable and restless and not taking his food. There was also present photophobia, typical whining cry, emaciation, redness of gums, hypotomia, sweating. In this instance there were rhonchi present in the chest and when the child scratched itself, a mark was left.

He had been exposed to the sun's rays. The question of diet was a difficult one. To insure sleep chloral hydrate had been given.

### Tumour of the Eyeball.

DR. E. O. MARKS showed a specimen of an eye. The child had contracted a mild iritis. The eye had been removed and on section of the specimen, a growth had been discovered. Radium had been used in the socket.

### Sarcoma of Eyelid.

DR. J. LOCKHART GIBSON showed a specimen of a tumour removed from behind the inner half of the upper eyelid. It had been of rapid growth and it had been regarded as a sarcoma. This view had been confirmed on microscopical examination.

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## THE MEDICAL BENEVOLENT ASSOCIATION OF NEW SOUTH WALES.

THE following reports of the Honorary Secretary and of the Honorary Treasurer of the Medical Benevolent Association of New South Wales for the year 1928 have been forwarded for publication.

### Honorary Secretary's Report.

The Treasurer's report shows that £315 was paid to beneficiaries out of ordinary revenue and £244 was carried forward to next year's account.

Administrative expenses amounted to less than £30, almost entirely for printing and postage.

A Christmas appeal issued in December met with a satisfactory response. The beneficiaries were very grateful for the money given them.

Although the actual result of the year's operations has been fairly satisfactory, yet it is a matter for deep regret that the great majority of the members of the profession in New South Wales are not supporting the Association.

THE MEDICAL BENEVOLENT ASSOCIATION OF NEW SOUTH WALES.

**Balance Sheet as at December 31, 1928.**

| LIABILITIES. | £ | s. | d. |
|---|---|---|---|
| Capital Account as at December 31, 1927 .. | 1,513 | 4 | 9 |
| Add Surplus for year ended December 31, 1928, as per Income and Expenditure Account .. .. .. .. .. .. .. .. | 244 | 13 | 11 |
| | £1,757 | 18 | 8 |

| ASSETS. | £ | s. | d. |
|---|---|---|---|
| Cash Accounts— | | | |
| Commercial Banking Co. .. .. .. .. | 93 | 8 | 4 |
| Government Savings Bank .. .. .. | 264 | 10 | 4 |
| Investments— | | | |
| N.S.W. Government Funded Stock .. .. | 900 | 0 | 0 |
| Commonwealth Inscribed Stock .. .. | 500 | 0 | 0 |
| | £1,757 | 18 | 8 |

**Income and Expenditure Account for Year Ended December 31, 1928.**

| EXPENDITURE. | £ | s. | d. |
|---|---|---|---|
| To Assistance to Beneficiaries .. .. .. .. | 315 | 0 | 0 |
| „ Christmas Gifts to Beneficiaries .. .. .. | 45 | 0 | 0 |
| „ Printing and Stationery .. .. .. .. | 14 | 2 | 0 |
| „ Stamps and Exchange .. .. .. .. .. | 13 | 3 | 3 |
| „ Balance Petty Cash .. .. .. .. .. | 1 | 16 | 9 |
| „ Bank Charge .. .. .. .. .. .. .. | 0 | 10 | 0 |
| „ Surplus for year added to Capital Account | 244 | 13 | 11 |
| | £634 | 5 | 11 |

| INCOME. | £ | s. | d. |
|---|---|---|---|
| By Donations .. .. .. .. .. .. .. .. | 65 | 7 | 6 |
| „ Annual Subscriptions (206) .. .. .. .. | 216 | 6 | 0 |
| „ Life Subscriptions (13) .. .. .. .. | 136 | 10 | 0 |
| „ Exchange added .. .. .. .. .. .. | 1 | 4 | 0 |
| „ Interest on Investments .. .. .. .. | 80 | 18 | 11 |
| „ Christmas Fund Appeal .. .. .. .. | 133 | 19 | 6 |
| | £634 | 5 | 11 |

M. O'GORMAN HUGHES, JOHN MACPHERSON } *Honorary Auditors.*
January 1, 1929.

E. S. LITTLEJOHN, *Honorary Treasurer.*

There are only 206 annual subscribers out of the 1,600 members of the British Medical Association in New South Wales. It should be borne in mind that the Association combines the principles of insurance with that of benevolence.

Every member of the Profession in New South Wales should become a member.

The Council is compelled to limit the assistance granted each beneficiary to what is just enough to support life. Much more could be done if the money were available. The lot of some of the widows is sad and pitiable enough. The Council also grants money for the support and education of the children. It is quite certain that the principle of insurance can be adapted to cover such cases as these. No one can be certain of what his future will be. Although a man may make reasonable provision for himself, how can he be sure that his children will never be in want? Poverty is bound to be the lot of some; but its incidence can be greatly lessened by such an organization as this.

The Council records with regret the death of Dr. S. H. MacCulloch who had recently succeeded Dr. Thomas Fiaschi as Chairman. Dr. MacCulloch took the greatest interest in the progress of the Association and helped it in every possible way. His loyal support and wise counsel will be greatly missed. Dr. A. W. Campbell was elected Chairman in his place and Dr. George Abbott was appointed to the vacant place on the Council.

### Honorary Treasurer's Report.

As the balance sheet shows, our income for the year was £634 and our expenditure £390, leaving a credit balance of £244. It must be remembered, however, that £88 of the balance belongs to the newly initiated Christmas Fund, so that the surplus for general purposes is only £156.

I regret to say that, instead of an advance, there has been a falling-off of three in the number of life subscriptions and ten in the annual subscriptions. There is still only one benefactor.

In December an appeal was made for donations for Christmas gifts for our beneficiaries and the response of £133 was so generous that we were able not only to present our beneficiaries with substantial and most welcome Christmas gifts, involving a sum of £45 in all, but to initiate a Christmas Fund, in which the balance of £88 was invested, to be drawn upon in the future.

We have to thank Mrs. George Hurst and also the Illawarra Medical Association, the Northern Districts Medical Association and the Northern Suburbs Medical Association for their generous donations.

We are now in urgent need of more funds in order to discharge our obligations in the coming year and earnestly appeal for more annual subscribers.

## Special Correspondence.

### LONDON LETTER.

BY OUR SPECIAL CORRESPONDENT.

### Local Government Bill and the Voluntary Hospitals.

THE voluntary hospital system in England is one which has grown slowly but surely during the passage of many years. It occupies a proud position in the regard of the people of this country and anything which tends to alter the basic principle of the organization, is viewed with suspicion. In a recent debate in the House of Lords on the Local Government Bill Lord Dawson of Penn spoke at some length on the future position of the voluntary hospitals as it appeared to him to be outlined in the bill. It seems that certain clauses of the bill give powers to local municipal councils to found general hospitals, entirely apart from the present Poor Law infirmaries, and it is not laid down that the councils must, only that they may, coopt members of the curative side of the medical profession. The official health services of this country are run by a small body of men who have specialized in preventive medicine, and by reason of the nature of that work are out of touch with the treatment of disease. Though Lord Dawson did not detract in any way from the useful and necessary work done by these practitioners, he emphasized that, as four-fifths of the medical profession in this country practise curative medicine, it is only reasonable that proper safeguards should be introduced into the bill insuring that this side of the profession is adequately represented on the bodies proposing to erect and run new general hospitals. It is fully realized that the hospital accommodation in this country is not adequate

to the demand for treatment and with the advance of knowledge in all branches of medicine and surgery it is becoming increasingly evident that only institutions can deal properly with disease. The various departments of medicine are no longer water-tight compartments and in almost every case of illness the knowledge and skill of many branches of science are required to achieve a satisfactory result. Briefly this means that the patient can no longer be treated suitably at home, but must be accommodated in an institution, that is a hospital, where only can be found the expensive equipment necessary for each specialty.

The knowledge required for the management of Poor Law infirmaries which are controlled by the local authorities and by those versed in preventive medicine, is certainly not sufficient for the management of ordinary general hospitals and it does not seem right or sensible, therefore, that, admirable as is the work done by these men, the founding and management of a general hospital with its highly complicated equipment and technique should be left entirely in their hands to the exclusion of the advice and help of those who have spent the larger portion of their working lives in close connexion with hospital duties. Lord Dawson advocated that the present voluntary hospital system should be utilized and extended and suggested that the closest cooperation between the Ministry of Health, local councils and the medical profession could be insured by the setting up under the chairmanship of the medical officer of health of an advisory council of doctors in each district and by this means bringing "the experience of the men who knew to the benefit of the medical men who had to administer."

### Radium and the London Hospitals.

Sir John Rose Bradford, President of the Royal College of Physicians, recently gave details of the methods by which the King Edward's Hospital Fund Committee propose to deal with the radium purchased for use in the London Hospitals with Sir Otto Beit's magnificent gift of £50,000. He explained that they first ascertained the amount of radium at present available in the hospitals, in some cases the hospital property and sometimes obtained on loan. It was then decided that four teaching hospitals should become radium centres, that is that these hospitals should receive from the fund a sufficient supply of radium which with their own supply should enable them not only to treat their own patients, but also be available for a supply of radium emanation to other hospitals. Besides these centres certain hospitals are to be allocated a supply and in making the distribution, many factors must be considered, such as the number of beds, the type of disease dealt with and so forth. The hospitals eligible to receive a supply of radium were asked to inform the King's Fund of the scheme they suggested for its use and the methods they proposed to adopt to insure the safety of the radium. The radium will be on loan to each hospital for one year and after that will be subject to withdrawal on one month's notice. It is laid down that the radium is to be available for all hospital patients, both ordinary and pay-bed, and that it must not be removed from the hospital without written permission from the fund authorities. The fund would insist on receiving regular reports from the hospital participating in the distribution as to the amount of radium available, the milligramme hours of its use and the number of patients treated. The president also emphasized the fact that in addition to the price of the radium itself, the cost of placing it in containers suitable for use in treatment is very high and this considerable item of expenditure has to be remembered in calculating the amount of radium that can be bought with any given sum.

In connexion with the purchase of radium it was recently pointed out that it is at present a monopoly and it is interesting to read that a well-known financier, Mr. E. W. Meyerstein, a member of the London Stock Exchange, has approached Mr. Robert Morrison, M.P., with a scheme by which it is hoped to obtain radium for the hospitals either as a free gift or at greatly reduced prices. It is suggested that a small company should be incorporated in England with the object of obtaining mines and producing radium. Any individual who subscribes a certain amount of stock will be allotted one founder's share and this on the same principle as obtains in regard to hospital subscribers will endow its owner with the right to nominate each year one hospital or institution for the free receipt of a stated amount of radium. It appears that in connexion with this suggested scheme an option has already been obtained on mines in Portugal from which radium has been produced.

Further details of this proposal which on the face of it seems an excellent one, will be awaited with much interest.

## Correspondence.

### "PROPHYLACTIC OBSTETRICAL PRACTICE."

SIR: Another Daniel, *alias* Belteshazzar, *alias* Kesteven has come to judgement:

> "All the presidents of the kingdom, the governors, and the princes, the counsellors, and the captains, have consulted together to establish a royal statute . . . that it be not changed, according to the law of the Medes and Persians, which altereth not."

Doctor "Daniel B. Kesteven" has flouted the law "which altereth not," and was promptly thrown into the "den of lions." In reply to anxious inquiries, he can be heard singing lustily, "O King, live for ever. . . O King, have I done no hurt." So the king showered presents and preferments upon him, because, as he said, the God of Daniel "delivereth and rescueth."

Whatever way we look at Dr. Kesteven's method of routine forceps delivery, he has proved in this series that the practice is not complicated by sepsis if the greatest possible care is exercised to rid his hands, gloves and instruments of septic infection from a previous case of any sort. His scathing comments on the ridiculously weak antiseptic (?) lotions in common use I receive with applause.

I am very pleased to have this further confirmation of the views expressed in my book, recently published, entitled "Lister Redivivus."

The late Baron Lister would not be surprised by the results shown as can be proved by a reference to his remarks on this subject which are very much to the point.

The discomfiture of the autoinfectionist is in keeping with my own views on the subject. It is evident that Dr. Kesteven is as skilful with the forceps as J. B. Hobbs is with the bat, knowing when to "pull" and where to "place." The record is an interesting one courageously told.

Yours, etc.,

A. C. F. HALFORD.

Wickham House, Brisbane,
January 5, 1929.

### COD LIVER OIL AND MUTTON BIRD OIL.

SIR: My attention was recently directed to the discovery by Professor Erik Agdur, of Uppsala, Sweden, of a toxic principle in cod liver oil as recorded in the *Acta Paeditrica Scandinavia*, Volume V, Fasciculus 3-4, Volume VI, Fasciculus 2, and Volume VII, Fasciculus 3-4. Studying the action of cod liver oil on calves, pigs, cats, dogs and rodents, microscopical changes were noted in the digestive tract, the parenchymatous organs and the skeletal system. Fatty and pigmentary degenerations as well as lime incrustations were found in the heart muscle. In some cases when the administration of oil was discontinued, pathological changes actually healed, but the more serious lesions led to extensive degeneration in spite of discontinuing the oil.

Agdur and Stenstrom demonstrated functional disturbances of the heart action due to lesions of the auriculoventricular bundle. Agdur describes typical changes of the heart muscle in two prematurely born infants who had been given cod liver oil as a prophylactic against rickets.

It will be remembered that Professor Mellanby (*The British Medical Journal*, May 24, 1924) gave a warning in regard to cod liver oil as follows:

> Care must be taken in giving the cod liver oil, as both children and adults can easily be upset when the doses are too large. I seldom give more than two drachms thrice daily to any patient. Larger doses may well be taken by some adults, but even so they will sometimes develope tachycardia and cardiac discomfort.

Agdur explicitly claims that distinct changes are produced even by the small doses recommended by Mellanby when the administration is continued for months.

Dr. Rolf Hallgreen, District Medical Officer, Hallevikstrand, Sweden, points out that the most disquieting point is that the experimental animals gain weight and appear to be healthy in spite of severe organic lesions, found only at the autopsy.

For centuries cod liver oil has been the household remedy among the fishing population of Norway and the Hebrides. It was introduced as a medicine over a hundred years ago. Following the discovery of cod liver oil to be so rich in vitamin *D* (the antirhachitic principle) and vitamin *A*, the preparation has been looked upon almost as a panacea against rickets and for the treatment of malnutrition. Cod liver oil has actually been boomed by the alluring phrase "Bottled Sunshine" (*vide* New Health Association publications).

Dr. Hallgreen in *Public Health*, February, 1929, suggests that whilst the bad effects may have so far passed unnoticed, possibly we owe some of the increasing numbers of heart failures in later life to well intentioned endeavours to prevent and cure rickets by too large doses of cod liver oil.

The practical conclusion of the recent investigation seems to be that cod liver oil, whilst an excellent source of vitamins *A* and *D*, should only occasionally be used as a supplement to ordinary diet. A teaspoonful a day or a teaspoonful a week should be ample to supply the necessary amount of vitamin. On no account should the doses recommended in the current pharmaceutical text books be given over a period of three months without very careful investigation.

As the first to direct attention to the therapeutic value of mutton bird oil (*Australasian Medical Gazette*, 1900, *The British Medical Journal*, 1918) it would be interesting to find out whether this substitute for cod liver oil, the analysis of which is similar to that of cod liver oil and which also contains a high iodine content, has also any of the toxic principle now alleged to be present in cod liver oil.

Twenty-nine years ago I published the results of successful experiments in feeding with mutton birds two Norwegians suffering from phthisis at Tawa Flat, New Zealand.

In 1913, whilst spending six weeks in Flinders Island and Cape Barren Island, Bass Straits, investigating an epidemic which incidentally I described as scarlet fever simulating measles, and which Dr. Whetter, fresh from the Mawson Expedition, without meeting or having any communication with me, described as measles simulating scarlet fever, I ascribed an increase of five pounds in weight to the diet of mutton birds.

Mr. Jeffcott, Research Biological Chemist, "Glaxo," Limited, on visiting Sydney in 1919, having read my note in *The British Medical Journal* of the previous year, called to see me with regard to the food value of mutton bird oil. He expressed the opinion, with which I agreed, that the increase in weight from feeding on mutton birds, was undoubtedly due to them being rich in vitamin *A* or the ease with which the natural fat of birds is digested.

The food of the short tailed petrel (mutton bird) (*Puffinus tenuirostris* Temm) consists of minute organisms similar to that of the cod which secretes the oil, said to be two hundred and fifty times richer in vitamin *A* than butter. Fish, like mammals, probably derive the whole of this vitamin *A* either directly or indirectly from the vegetable kingdom, in their case from minute vegetable marine organisms, such as diatoms and algæ containing this vitamin, which form the food of myriads of minute marine animalculæ which in their turn are eaten by kinds of small fish, whose name is legion, and which are eaten by the cod and other fish or by the Antarctic-living migratory mutton bird. Other fish oils and especially whale oil also contain vitamin *A*, which probably accounted for the benefit some of the Australian Imperial Forces obtained from eating the frozen whale oil served out for smearing and rubbing the feet in the trenches in the 1916-1917 winter in Flanders.

Yours, etc.,

J. S. PURDY, M.D.,
Metropolitan Medical Officer of Health.

Queen Victoria Building,
Sydney.
March 22, 1929.

# Congress Notes.

## AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

### Papers at Meetings of Sections.

THE attention of members intending to contribute papers is directed to the rules laid down by the Federal Committee that notice of all papers and exhibits to be presented at any meeting of a section shall be received by the honorary secretaries or by the honorary secretary of the section not less than sixty days before the inaugural meeting of the session and that a complete typewritten copy of the papers shall be in the hands of the honorary secretary of the section not less than thirty days before the date of the inaugural meeting. Members are thus required to give notice of their intention to read papers not later than July 4, 1929, and to place their papers in the hands of the secretaries of sections not later than August 3, 1929. It is understood that members will keep a copy for their own use.

The secretaries of the sections are anxious to complete their programmes in detail without further delay. All intending contributors are urged to communicate with them as soon as possible. The work of the secretaries will be greatly facilitated if members intending to offer papers will intimate the fact to the secretary of the appropriate section without delay.

### Congress Ball.

Arrangements are being made for a Congress ball to be held on Friday evening, September 6.

### Congress Dinner.

The Congress dinner will be held on Wednesday, September 4, 1929, at half past seven in the evening. His Excellency Sir Dudley de Chair, Governor of New South Wales, will be present.

### Office-Bearers of Sections.

The following is a list of the Sections of Congress with their office-bearers:

*Section of Anæsthetics.*

*President*, Dr. Gilbert Brown, Gilberton, South Australia. *Vice-Presidents*, Dr. M. C. Lidwill, Sydney, New South Wales; Dr. G. F. V. Anson,[1] Wellington, New Zealand; Dr. E. Murphy, Brisbane, Queensland; Dr. F. W. Green, Kooyong, Victoria; Dr. M. K. Moss, Perth, Western Australia. *Secretary*, Dr. J. G. Hunter, Robey Street, Mascot, Sydney.

*Section of Dermatology.*

*President*, Dr. R. R. Wettenhall. Melbourne, Victoria. *Vice-Presidents*, Dr. Langloh P. Johnson, Sydney, New South Wales; Dr. F. H. Makin,[1] Adelaide, South Australia; Dr. J. E. McGlashan, Perth, Western Australia; Dr. H. W. Wilson, Auckland, New Zealand. *Secretary*, Dr. G. R. Hamilton, 147, Macquarie Street, Sydney.

The sign [1] indicates that a reply to the invitation has not yet been received.

*Section of Medical Literature and History.*

*President*, Dr. A. A. Lendon, North Adelaide, South Australia. *Vice-Presidents*, Dr. R. Scot Skirving, Sydney, New South Wales; Dr. J. Young, Invercargill, New Zealand; Dr. A. V. M. Anderson, Melbourne, Victoria; Dr. E. Sandford Jackson, Brisbane, Queensland; Dr. A. Webster, Perth, Western Australia. *Secretary*, Dr. L. Cowlishaw, Gordon Road, Lindfield, Sydney.

*Section of Medicine.*

*President*, Dr. C. T. Charles de Crespigny, Adelaide, South Australia. *Vice-Presidents*, Professor A. E. Mills, Sydney, New South Wales; Professor D. W. Carmalt Jones, Dunedin, New Zealand; Dr. Eustace Russell, Brisbane, Queensland; Dr. L. S. Latham, Melbourne, Victoria; Dr. W. H. Nelson, Subiaco, Western Australia. *Secretary*, Dr. Cotter Harvey, 235, Macquarie Street, Sydney.

*Section of Naval, Military and Air Medicine and Surgery.*

*President*, Dr. R. M. Downes, Melbourne, Victoria. *Vice-Presidents*, Dr. F. A. Maguire, Sydney, New South Wales; Dr. P. C. Fenwick, Christchurch, New Zealand; Dr. A. H. Marks, Brisbane, Queensland; Dr. H. H. E. Russell, Unley, South Australia; Dr. W. W. Giblin,[1] Hobart, Tasmania; Dr. D. M. McWhae, Perth, Western Australia. *Secretary*, Dr. R. W. W. Walsh, Everton Road, Strathfield, Sydney.

*Section of Neurology and Psychiatry.*

*President*, Dr. W. E. Jones, Melbourne, Victoria. *Vice-Presidents*, Dr. C. A. Hogg, Sydney, New South Wales; Dr. W. M. Macdonald, Dunedin, New Zealand; Dr. J. P. Lowson, Brisbane, Queensland, Professor H. H. Woollard,[1] Adelaide, South Australia; Dr. E. A. Rogers, Hobart, Tasmania; Dr. J. Bentley, Claremont, Western Australia. *Secretaries*, Dr. R. A. Noble, 143, Macquarie Street, Sydney; Dr. J. A. L. Wallace, Callan Park, New South Wales.

*Section of Obstetrics and Gynæcology.*

*President*, Dr. H. Jellett, Christchurch, New Zealand. *Vice-Presidents*, Dr. R. L. Davies, Sydney, New South Wales; Dr. F. A. H. Michôd, Brisbane, Queensland; Dr. T. G. Wilson, North Adelaide, South Australia; Dr. R. H. Morrison, Melbourne, Victoria; Dr. J. K. Couch, Perth, Western Australia. *Secretary*, Dr. A. J. Gibson, 143, Macquarie Street, Sydney.

*Section of Ophthalmology.*

*President*, Dr. D. D. Paton, Perth, Western Australia. *Vice-Presidents*, Dr. R. H. Jones, Sydney, New South Wales; Dr. W. A. Fairclough, Auckland, New Zealand; Dr. G. Thomson, Brisbane, Queensland; Dr. H. F. Shorney, Adelaide, South Australia; Dr. G. H. Hogg, Launceston, Tasmania; Dr. L. Mitchell, Melbourne, Victoria. *Secretary*, Dr. W. M. C. Macdonald, 235, Macquarie Street, Sydney.

*Section of Orthopædics.*

*President*, Dr. A. V. Meehan, Brisbane, Queensland. *Vice-Presidents*, Dr. R. B. Wade, Sydney, New South Wales; Dr. D. S. Wylie, Palmerston North, New Zealand; Dr. H. Gilbert, Adelaide, South Australia; Dr. C. G. Shaw, Melbourne, Victoria; Dr. A. Juett,[1] Perth, Western Australia. *Secretary*, Dr. J. Hoets, 143, Macquarie Street, Sydney.

*Section of Oto-Rhino-Laryngology.*

*President*, Dr. R. Graham Brown, Brisbane, Queensland. *Vice-Presidents*, Dr. R. S. Godsall, Sydney, New South Wales; Dr. J. Hardie Neil, Auckland, New Zealand; Dr. H. M. Jay, Adelaide, South Australia; Dr. J. S. Barr,[1] Hobart, Tasmania; Dr. G. A. D. McArthur, Melbourne, Victoria. *Secretary*, Dr. Garnet Halloran, 143, Macquarie Street, Sydney.

*Section of Pædiatrics.*

*President*, Dr. S. W. Ferguson, Melbourne, Victoria. *Vice-Presidents*, Dr. E. H. M. Stephen, Sydney, New South Wales; Dr. G. B. Sweet, Auckland, New Zealand; Dr. T. H. R. Mathewson, Brisbane, Queensland; Dr F. N. le Messurier, Adelaide, South Australia; Dr. A. W. Shugg, Hobart, Tasmania; Dr. R. H. Crisp,[1] Perth, Western Australia. *Secretary*, Dr. M. J. Plomley, 233, Macquarie Street, Sydney.

*Section of Pathology and Bacteriology.*

*President*, Professor P. MacCallum, Melbourne, Victoria. *Vice-Presidents*, Dr. Keith Inglis, Drummoyne, New South Wales; Dr. N. M. Gutteridge, Toowoomba, Queensland; Dr. J. H. B. Walsh, Hobart, Tasmania; Dr. B. C. Cohen, Perth, Western Australia. *Secretary*, Dr. F. S. Hansman, 143, Macquarie Street, Sydney.

*Section of Radiology and Medical Electricity.*

*President*, Dr. H. M. Hewlett, Melbourne, Victoria. *Vice-Presidents*, Dr. W. B. Dight, Sydney, New South Wales; Dr. P. D. Cameron, Wellington, New Zealand; Dr. B. L. W. Clarke, Brisbane, Queensland; Dr. J. Stanley Verco, Adelaide, South Australia; Dr. W. P. Holman, Launceston, Tasmania; Dr. A. Syme Johnson, Perth, Western Australia. *Secretary*, Dr. H. M. Cutler, 139, Macquarie Street, Sydney.

*Section of Surgery.*

*President*, Professor Gordon Bell, Dunedin, New Zealand. *Vice-Presidents*, Dr. G. H. Abbott, Sydney, New South Wales; Dr. D. S. Wylie, Palmerston North, New Zealand; Dr. G. P. Dixon, Brisbane, Queensland; Dr. B. Smeaton, Adelaide, South Australia; Dr. F. W. Fay, Hobart, Tasmania; Dr. B. T. Zwar, Melbourne, Victoria; Dr. F. A. Hadley, Perth, Western Australia. *Secretary*, Dr. T. Farranridge, 229, Macquarie Street, Sydney.

In one or two instances final arrangements have not been made.

### Time Table.

In our issue of April 27, 1929, page 573, an error has been made in the arrangements for Monday, September 2, 1929. The registration of members of Congress will take place in the morning and not in the afternoon as stated.

### Museum Committee.

The Honorary Secretary of the Museum Committee, Dr. Keith Inglis, Wolseley Street, Drummoyne, announces that his committee proposes to arrange an exhibit in the museum illustrating the hobbies, pursuits and collecting habits of medical men when not engaged in their ordinary professional work. It is known that certain members of the medical profession engage in such pastimes as cabinet making, entomology, botany and the like. An invitation is extended to these practitioners to contribute an exhibit of their work for the museum of the Congress. In addition collections of articles of interest, such as antique surgical instruments, would be acceptable.

Intending exhibitors are requested to notify the Honorary Secretary of the Museum Committee, giving particulars of the nature of the proposed exhibit, its size and the space required for display.

## Obituary.

### THOMAS BENNETT WALLEY.

WE regret to announce the death of Dr. Thomas Bennett Walley which occurred at Sydney on April 29, 1929.

### THE LATE GEORGE ADLINGTON SYME.

IT is proposed to hold a memorial service to George Adlington Syme at the Medical Society Hall, East Melbourne, on Sunday, May 12, 1929, at 2.30 in the afternoon. Speeches will be delivered by selected leaders of the medical profession in the several States. In view of this service the publication in this journal of a record of his career and an appreciation of the man and his life will be postponed in order that these may appear simultaneously with the speeches that will be made.

# Proceedings of the Australian Medical Boards.

## QUEENSLAND.

The undermentioned have been registered under the provisions of *The Medical Act of* 1925, of Queensland, as duly qualified medical practitioners:

Stanley, George Percival, M.B., Ch.M., 1891 (Univ. Sydney), Dunwich.

Restoration to the Register:

Tipping, Frank, M.B., 1907 (Univ. Melbourne), Chillagoe.

Registration of additional qualification:

O'Brien, Daniel Patrick, M.B., Ch.B., B.A.O., N.U.I., 1911, Rockhampton.

## THE EDWARD HENRY EMBLEY MEMORIAL.

WE have been requested to announce by the Edward Henry Embley Memorial Committee that the following subscriptions have been received by the Honorary Treasurer:

T. F. Ryan, £5 5s. 6d.

Edward Ryan, £3 3s.

Miss Alicia Burton, F. L. Davies, E. L. Gault, H. J. Gray, H. M. Hewlett, J. Leon Jona, L. S. Latham, H. Laurie, Fay Maclure, Alan Newton, H. H. Turnbull, Mary Agnes Wait, A. M. Wilson, £2 2s. each.

M. K. Moss, £1 2s.

F. W. Grutzner, N. R. Henderson, £1 1s. 6d. each.

J. M. Andrew, John Bristow, R. C. Brown, Leo Doyle, D. M. Embelton, S. A. Ewing, J. W. Florance, F. W. Green, W. R. Groves, G. B. Halford, L. E. Hurley, J. Jona, F. B. Lawton, F. R. Legge, D. McLean, D. M. Morton, Hilda Rennie, E. R. Sawrey, G. C. Scantlebury, Mrs. R. Gray Smith, £1 1s. each.

C. Thorogood, £1.

Mary De Garis, A. Steven, 10s. 6d. each.

# Diary for the Month.

MAY 14.—Tasmanian Branch, B.M.A.: Branch.
MAY 14.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 15.—Western Australian Branch, B.M.A.: Branch.
MAY 16.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 21.—Tasmanian Branch, B.M.A.: Council.
MAY 21.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAY 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 22.—Victorian Branch, B.M.A.: Council.
MAY 24.—Queensland Branch, B.M.A.: Council.
MAY 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 28.—Illawarra Suburbs Medical Association, New South Wales.
MAY 30.—New South Wales Branch, B.M.A.: Branch.
MAY 30.—South Australian Branch, B.M.A.: Branch.

# Medical Appointments Vacant, etc.

**For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page XX.**

COMMONWEALTH DEPARTMENT OF HEALTH: Medical Officer.
COMMONWEALTH REPATRIATION COMMISSION: Medical Officer.
PUBLIC SERVICE COMMISSION (VICTORIA): Medical Officer.
ROYAL HOSPITAL FOR WOMEN, PADDINGTON: Resident Medical Officer.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY: Senior Resident Medical Officer, Junior Resident Medical Officer.

# Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney. | Australian Natives' Association.<br>Ashfield and District United Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham United Friendly Societies' Dispensary.<br>Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney.<br>North Sydney Friendly Societies' Dispensary Limited.<br>People's Prudential Assurance Company, Limited.<br>Phœnix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing.<br>Brisbane United Friendly Society Institute.<br>Stannary Hills Hospital.<br>Boonah District Hospital. |
| SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide. | All Contract Practice Appointments in South Australia.<br>Booleroo Centre Medical Club. |
| WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia. |
| NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

# Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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